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INTERATRIAL SEPTAL DEFECTS*

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Atrial septal defects occur as the result of abnormal development or incomplete fusion of one or more of the septal constituents. The 3 varieties arise as follows:

1. The septum primum grows down from the dorso-cephalic wall of the common atrium to join the upper margin of the atrioventricular cushions which have grown in from the common atrioventricular ring to divide it into tricuspid and mitral valves and themselves to fuse with the inter-ventricular septum. Incomplete development of the septum primum will result in the 'ostium primum' type of inter-atrial septal defect, while any retardation of the growth of the atrioventricular cushions will produce a partial or complete form of atrioventricularis communis.

2. An ostium secundum develops as a fenestration of the septum primum. This is generally closed by the septum secundum, which grows as a curtain over the ostium. Abnormal development of the septum secundum will result in the ostium secundum remaining patent.

3. The 'sinus venosus' defect is the result of abnormal development of the sinus venosus and lies close to the superior-vena-caval orifice.

* Paper presented at the 42nd South African Medical Congress (M.A.S.A.), East London, C.P., September - October 1959.

Morbid Anatomy of Atrial Septal Defects

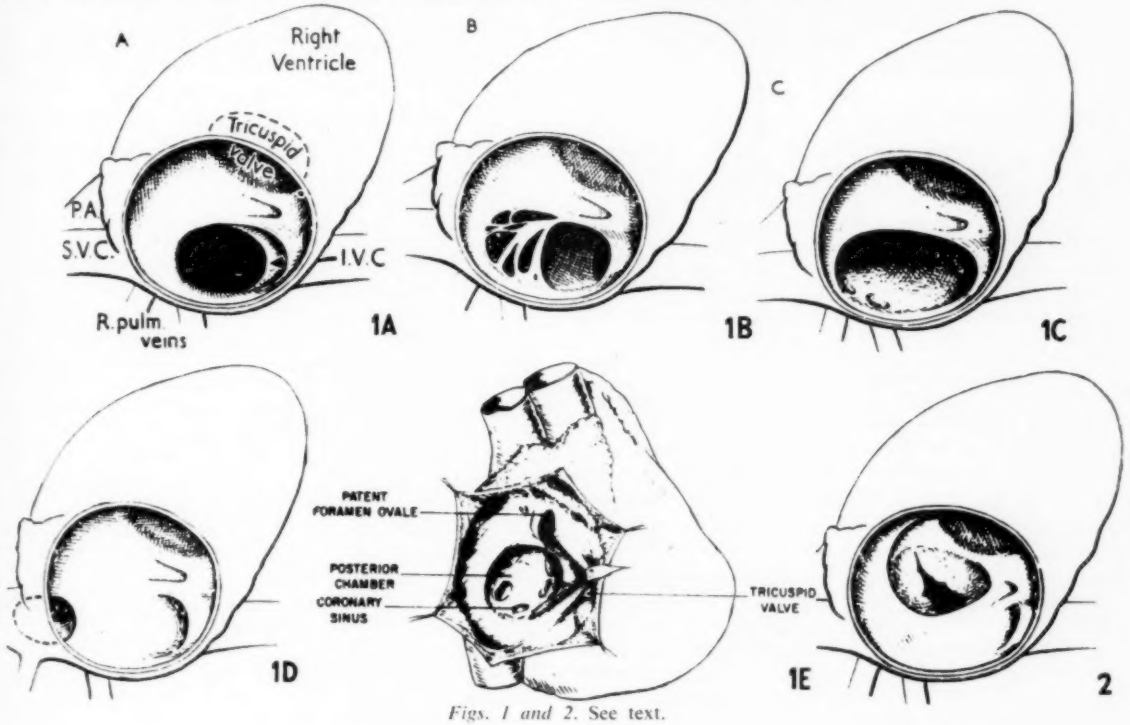
1. 'Septum Secundum' Defects, with which are Included Defects in the Foramen Ovale

(*a*) Partial defect in the valve of the foramen ovale, which may be represented by a fenestrated membrane (Fig. 1 B).

(*b*) Complete absence of the valve of the foramen ovale. In this the lower margin may be continuous with the valve of the inferior vena cava and anomalous pulmonary venous drainage is a common association (Figs. 1 A and 1 C).

(*c*) 'Sinus venosus'-type of defects. These are situated high in the septum immediately caudal to the entrance of the superior vena cava and frequently have anomalous veins from the right lung draining into the right atrium or even into the right superior vena cava (Fig. 1 D). The venous drainage from the left lung may return to a persistent left superior vena cava or to the coronary sinus.

(*d*) Combined defects, perhaps with total anomalous venous drainage (Fig. 1 E).



Figs. 1 and 2. See text.

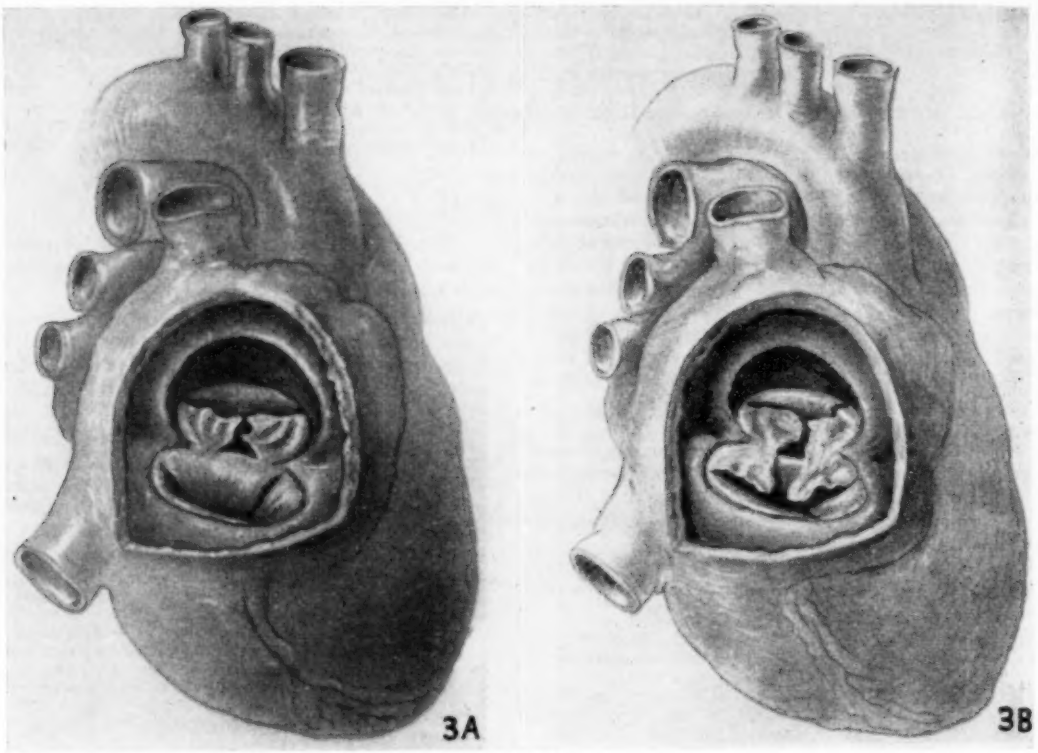


Fig. 3. See text.

2. *'Septum Primum' Defects*

These are less common. They are situated in the lower portion of the septum in front of the orifice of the coronary sinus and their inferior rim is the upper edge of the ventricular septum (Fig. 2). In some classifications these are included as a sub-group of the following category.

Of surgical significance in this group is the proximity of the AV conducting bundle to the posterior free edge of the defect.

3. *Atrioventricularis Communis*

(*a*) Partial. The interventricular septum is intact but, associated with the 'ostium primum' defect, there is a cleft or defect in the septal cusp of either or both mitral and tricuspid valves (Figs. 3 A and 3 B).

(*b*) Complete. There is a defect of the upper portion of the ventricular septum as well.

Associated Lesions

Atrial septal defects of the 'secundum' type often occur as isolated phenomena. The association of atrial septal defect with the tall asthenic build, high arched palate, and arachnodactyly, is too well known to elaborate further. A patent foramen ovale may be of no haemodynamic significance unless an accompanying obstruction to the pulmonary outflow causes raised right atrial pressure and the shunting of desaturated blood to the left atrium. As the fifth component of the pentalogy of Fallot it has no great significance symptomatically but naturally adds to the complexity of the repair. From case reports in the literature the association with mitral stenosis (Lutembacher's syndrome) would appear to be uncommon. Simultaneous separate defects in atrial and ventricular septa occur frequently; so do associated anomalous pulmonary veins. Indeed any congenital cardiovascular abnormality may have an associated interatrial defect.

The 'primum' type of defect, by its very nature, is often associated with abnormalities in the development of both mitral and tricuspid valves as well as of the ventricular septum.

Symptomatology and Diagnosis

It is not the purpose of this communication to discuss either symptomatology or diagnosis.* Suffice it to say that in the neonatal period atrial septal defect occurring alone may produce severe cardiac symptoms and lead to early death, but it is more usual for the secundum defect to remain undetected for a number of years, although there may be radiological evidence of considerable cardiomegaly. In spite of being asymptomatic it is noticeable how many of these patients have had no inclination to participate in strenuous activities. Progressive symptoms of disability generally commence in the third decade and continue for several years, until a severe degree of pulmonary hypertension destroys any chance of effective

* See article by Drs. Zion, Bradlow and Braudo at page 810 of this issue.

TABLE I. RESULTS OF OPERATIVE TREATMENT IN THE FIRST 23 CASES

Case	Age	Sex	Date	Method	Defect	Pressures RA	Pressures RV	Pressures PA	Result	Comment
1.	9	M	3.2.58	Hypothermia	Secundum + anom. R. pulm. veins	5	54/4	44/12	Cured	See Table II
2.	14	F	4.6.58	Hypothermia	Secundum	—	28/7	—	Cured	
3.	27	F	22.8.58	Pump	Secundum (*a*)	—	—	—	Good	Portion of defect not closed
4.	39	F	9.9.58	Pump	Secundum (*b*)	—	40/0	45/20	Died	Postop. haemorrhage (*e*)
5.	9	F	18.9.58	Pump	Secundum + anom. veins (*c*)	8/2	55/0	55/22	Persistent hypertension (*f*)	
6.	11	F	29.10.58	Pump	Secundum + MI	8(*d*)	56/20	56/42	Cured	
7.	19	F	4.11.58	Hypothermia	Secundum	6/0	34/5	28/5	Cured	
8.	2	M	12.11.58	Pump	Total anom. pulm. veins	8/2	54/0	45/11	Died	Postop. haemorrhage (surg.)
9.	2	M	31.12.58	Hypothermia	Secundum + VSD	11/4	60/25	65/35	Improved	VSD not closed
10.	9	F	7.1.59	Pump	Trilogy	11	185/15	15/8	Cured	
11.	31	F	17.1.59	Hypothermia	Secundum	8/3	29/6	25/9	Cured	
12.	5	F	13.2.59	Hypothermia	Secundum	5/0	25/1	23/8	Cured	
13.	45	F	20.3.59	Hypothermia	Secundum	8/-	40/2	28/12	Cured	
14.	22	F	13.3.59	Hypothermia	Secundum	15/4	100/10	102/50	Died (*g*)	(*h*)
15.	10	M	16.4.59	Pump	AV communis	7/3	35/1	35/15	Died	Haemolysis (*i*)
16.	16	M	28.4.59	Hypothermia	Secundum	—	—	35/15	Cured	
17.	19	F	6.5.59	Hypothermia	Secundum	9/3	50/0	40/15	Cured	
18.	4	F	12.6.59	Hypothermia	Secundum +	6	40/2	45/22	Cured	
19.	9	M	6.8.59	Pump	Trilogy	2	100/12	44/27	Cured	
20.	4	F	20.8.59	Pump	AV communis	10/5	—	50/25	Cured	
21.	16	F	25.8.59	Hypothermia	Secundum	6	30/5	26/12	Cured	
22.	16	F	8.9.59	Hypothermia	Secundum	5/3	42/0	42/10	Cured	
23.	21	F	10.9.59	Pump	Trilogy	10/8	125/4	18/11	Cured	

RA = right atrium. RV = right ventricle. PA = pulmonary artery. MI = mitral incompetence. VSD = ventricular septal defect. (*a*) 4 litres L - R shunt. (*b*) Catheter 1954. (*c*) Catheter 1955 (see Table III). (*d*) Clean pressure. (*e*) Christmas factor. (*f*) See Table III. (*g*) Died suddenly 12 hrs. postop. (*h*) 6 months pregnant. Pulm. artery pressure greater than systemic. See text. (*i*) Too high line-pressure during bypass.

relief. Associated cardiac abnormalities naturally make the symptoms and prognosis much worse.

Natural History of Atrial Septal Defect

During foetal life the normal blood flow passes from right to left through the foramen ovale. After birth the increased pressure in the left atrium closes the valve of the foramen ovale and prevents left-to-right flow. However, if a large defect is present the two ventricles fill from a common chamber and the quantity of blood entering each ventricle depends upon the resistance to filling which each offers. At birth the two ventricles are of equal muscular development and the pressures in the pulmonary and systemic circuits are about the same. With the physiological fall in pulmonary vascular resistance and rise in the systemic resistance more blood passes through the right side than the left. At first the right ventricle dilates and the pulmonary vascular bed increases, but as time passes secondary changes occur in the pulmonary vasculature. This takes time and generally in early life few symptoms develop. As the degree of left-to-right shunt increases, enormous amounts of blood may pass through the pulmonary circuit and perhaps only one-tenth as much passes through the systemic. With the development of pulmonary arteriolar changes the amount of this shunt diminishes and ultimately, in the extreme case with severe pulmonary hypertension, the pressure in the pulmonary artery may actually exceed that in the aorta and blood may shunt in the reverse direction through the defect (see case 14, Table I). The ages at which these changes occur cannot be predicted. In some infants in whom the normal foetal pulmonary vascular resistance persists, death may occur at an early age from congestive cardiac failure. More commonly symptoms develop slowly and may not be severe until the 2nd or 3rd decade. There are some instances of patients living out a normal life span with relative ease, while there are many instances of severe secondary pulmonary hypertension being encountered under the age of 10 years. Swan[2] reports, 'We have seen patients pass from the first group to the second in a period as short as a year or two' (Table III). The significance of this lies in the graver prognosis in the second group and in the possibility that permanent obliterative vascular changes will militate against the beneficial effect that surgery may otherwise have offered. The mean life expectancy is not much over 37 or 38 years.

SURGERY

Assessment for Surgery

The first requirement is adequate diagnosis. In Johannesburg we are fortunate in having the excellent diagnostic facilities and clinical acumen of a first-class privately-run Cardiac and Catheter Unit, as well as the Cardiac Clinic of the Johannesburg General Hospital. These groups of clinicians are able to present the surgeon with a thoroughly prepared assessment of the patient. This includes clinical history, physical signs, ECG, phonocardiogram, and radiology. Final confirmation is obtained from cardiac catheterization when the right heart chambers have been thoroughly explored with the catheter, where anomalous pulmonary venous drainage can be detected

TABLE II. CATHETER DATA IN CASE 1 (J.R., AGED 9, OPERATION 3.2.58)

	Pre-operative		*Postoperative*	
Site	*mm.Hg*	*Oxymetry*	*mm.Hg*	*Oxymetry*
Main PA	44/12	90.5%	28/10	71%
RV	54/4	90.2%	28/2	70%
Mid RA	5	92.5%	4	71%
Low RA		68.0%		65%
Peripheral flow—1·45 litres per minute Pulmonary flow—7·8 litres per minute			No evidence of left-to-right shunt	

TABLE III. CATHETER DATA IN CASE 5 (V.S., AGED 9, OPERATION 18.9.58)

	Pre-operative (8.7.55)		*Postoperative (2.7.59)*	
Site	*mm.Hg*	*Oxymetry*	*mm.Hg*	*Oxymetry*
Main PA	50/22	76%	70/50	56%
RV	55/0	78%	70/5	56%
Mid RA	8/2		8	56%
Low RA		80%		58%
Brachial artery			100/70	

with a high degree of certainty and where shunts can be picked up and pressures recorded. Other associated anomalies are confirmed or excluded. Should circumstances require it, selective angiocardiography can be performed. The end-result of these investigations is the estimation of the flow across the defect, the pulmonary vascular resistance and the degree of pulmonary hypertension, if any.

The amount of flow and degree of pulmonary hypertension are of prime importance. A large left-to-right shunt is an encouraging finding and is an indication of increased flow through the lungs, while a raised pulmonary vascular resistance diminishes this flow and is a pointer to probable structural pulmonary vascular changes. So important are these changes that, when they become severe, corrective surgery is unlikely to succeed and, indeed, may be dangerous. Dammann and Ferencz,[3] in a study of hypertensive vascular disease complicating congenital cardiac anomalies, state: 'We do not know when, in the course of the disease, pulmonary vascular changes become so severe that operation is no longer indicated'.

It is because we do not know when pulmonary vascular changes may develop that surgery is recommended in an early phase; it should not be postponed until symptoms progress, as is so often done. When the vascular changes have passed from the phase of intimal fibrosis associated with medial hypertrophy to one of dilatation, as is found in grade-4 and grade-5 pulmonary hypertension, then closure of the defect is unlikely to result in lowering of the pulmonary blood pressure. In the Mayo Clinic series of cases with hypertension, the ratio of pulmonary systolic pressure to systemic pressure rose in all of the grade-5 cases after closure of the defect.[4] It is also stated from the Mayo Clinic: 'Analysis of accumulated experience with the repair of atrial septal defect in 119 adults has revealed several factors determinable pre-operatively that appear to have a strong influence on the operative risk. These factors are related to the presence of pulmonary vascular disease or heart failure and contribute importantly to the selection of patients for operation'.[5] They also found that if congestive heart failure had occurred the mortality was 39%, whereas in its absence the rate was 7%. Patients whose right atrial pressure reached a peak of 15 mm.Hg or more had a 50% hospital mortality; so did patients with a right-to-left shunt of 10% or more. They list 5 factors in determining the influence of abnormal values on the outcome of an operation, viz. (*a*) congestive cardiac failure, (*b*) markedly elevated right atrial pressure, (*c*) a large right-to-left shunt, (*d*) severely elevated pressure in the pulmonary artery, and (*e*) markedly increased pulmonary vascular resistance.[5] If none of these unfavourable factors, or only one of them, exists, the risk of operation is well acceptable. The risk is high if 2 of them are present, and extreme if 3 or more coexist.

It is generally accepted that a diagnosis of an uncomplicated lesion in patients under 40 years of age is sufficient to be a firm indication for surgery irrespective of whether symptoms are present or not.

In Swan's series[2] of over 100 cases only 1 death occurred in a patient under the age of 18 years, while Kirklin[6] reported no death under the age of 20. These figures are comparable with those of Holmes Sellors.[7] In view of these low mortality figures the decision is not *whether* surgery should be advised, but *when*. In the absence of clear contra-indications surgery should be advised in all cases. Swan[2] has stated that when pulmonary blood flow is no more than 1 litre per minute higher than the systemic, serious doubts about the advisability of surgery should be entertained. Unfortunately, when such a problem arises, knowing that no other treatment will help the patient, the surgeon is often persuaded to operate in the hope of affording some measure of relief; and more often than not this does not materialize (see case 14, Table I).

Operative Techniques

As with most surgical procedures, the techniques have passed through varied evolutionary phases. Older and cruder methods have given way to the present direct approach. It will serve no purpose to enumerate the many earlier types of operation. At present it is almost universally accepted that direct visualization of the defect with occlusion of the venous inflow is the most desirable method. This can only suffice under conditions of hypothermia or with the use of the pump-oxygenator. The first closure of an atrial septal defect by direct suture in a bloodless field was performed by Lewis, using hypothermia, in 1953. Since that time many thousands of such defects have been closed successfully, with a mortality in the good-risk patient of about 1%.

Hypothermia has the advantage of simplicity and does not require the elaborate set-up that is used with a pump oxygenator. It has, however, the great disadvantage that the surgeon is gravely limited in time. At the usual tempe-

ratures employed, 6 - 8 minutes of circulatory occlusion is all the time available. This may not allow of the more complicated defects, or associated defects, being effectively cured. Nevertheless in the usual 'secundum' type of interatrial defect, which comprise about 90% of such defects, the allotted time is quite adequate for complete closure. If need be, after waiting a sufficient time to re-establish the circulation, the surgeon may make a second entry.

Pump oxygenation should be employed in all cases where the diagnosis may be in doubt, in all cases of 'ostium primum' or 'atrioventricularis communis' defect, and where other abnormalities coexist. It cannot be denied that this method may be the ultimate ideal in all cases of interatrial defect. However, for two reasons we have not been using it, thus: Firstly, where the diagnosis is not in doubt, even though one has to work against time, direct closure under hypothermia can be effected completely and with small risk.[2,7] Secondly, with our present available opportunities, where we have a long waiting list of more complicated conditions, precedence with the pump must be given to these. Possibly if we are able to eliminate our present backlog of cases (not a very likely prospect) we may change. We have indeed operated upon 3 pure secundum defects using the pump, but this was early on when we chose to operate upon uncomplicated abnormalities. Two further cases in whom the diagnosis was in doubt have been operated on using the pump.

Results of Operative Treatment

The first case of atrial septal defect was operated upon under hypothermia on 3 February 1958. Since then 27 more cases have been operated upon under hypothermia and 12 with pump oxygenation.

Two cases have been catheterized after operation and their pre- and postoperative findings are presented (Tables II and III). These two cases illustrate dramatically the complete restoration to normality in J.R. and the persistence of pulmonary hypertension in V.S., whose condition was allowed to persist too long before surgical correction.

SUMMARY

Twenty-eight cases of secundum defect have been operated upon under hypothermia, 1 of whom has subsequently been found to have an associated ventricular septal defect. In 13 cases the defect has been closed with the aid of the pump oxygenator — 5 of these having uncomplicated secundum defect; 1, secundum defect associated with rheumatic mitral incompetence, which was corrected at the same time; 3, secundum defects associated with pulmonary valvular stenosis; 3, atrioventricularis communis; and 1, total anomalous pulmonary venous return.

Among the pure secundum defects 2 patients have died, one under hypothermia, the other with the pump. The first (case 4, Table I), who died a haemorrhagic death[8] after being adequately repaired on the pump, was a 39-year-old woman who had pulmonary hypertension and severe disability, could climb only 8 steps, and had been having haemoptysis for 6 months. The other was a 22-year-old woman, 7 months pregnant, weighing 78 lb, who had an extreme degree of pulmonary hypertension, the pulmonary-artery pressure being 10 mm.Hg higher than the systemic, with a left-to-right flow of half a litre per minute; in the light of present knowledge she should not have been operated upon.

Of the 3 cases of atrioventricularis communis, 1 died of a technical fault during the bypass phase of the pump. The child with total anomalous venous drainage died from a technical fault connected with the surgery. These 2 deaths should have been avoided. However, in cases of pulmonary hypertension high mortality must be expected if the operation is performed.

Acknowledgements are due to Mr. D. Adler, who operated on half of these patients, and to our large team of medical and nursing colleagues, who have been essential to our work.

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WHITHER OPHTHALMOLOGY ?*

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I have chosen as the title of this paper 'Whither ophthalmology?', because I feel that we have reached a stage here in Johannesburg, and possibly in other centres of the Union, where some reorganization is necessary if we are to face the future with a definite plan.

This facet of medicine, although only concerning a small part of the body, has become so vast that it is impossible for anyone to be expert in all its ramifications, and has led to what one might call 'specialists within a specialty'. It has therefore become advisable for these 'specialists' to practise as a team, and I am of the opinion, and I know that many of you are too, that the best way in which such an effort could be organized is by having a central eye hospital. Thus the ophthalmologists who are already concerning themselves with one particular branch of this specialty could unite their efforts. This hospital should have at its head an ophthalmologist-director.

A teaching school should be established, where first of all the general practitioner of ophthalmology could be trained to give a service to the people in the outlying districts which they are not getting now, nor will get in the foreseeable future, unless some plan is made to increase the number of ophthalmologists.

Before starting on such a hospital, one has, of course, to consider what accommodation would be required. At the moment about 30 beds are being occupied at the General Hospital, approximately 10 - 15 at the Childrens' Hospital, and about 50 beds in nursing homes. Therefore, a hospital having about 100 beds would probably be a minimum requirement, with provision for extension at any time. It would, of course, cater for patients of all income brackets. Apart from the bed accommodation, many other departments would have to be accommodated, such as:

1. A *detachment unit*, where all detachments could be referred for further investigation and adequate treatment. It is obvious that a doctor treating detachments daily would be more expert than one who only occasionally saw and treated

* Address delivered by the retiring Chairman at the Annual General Meeting of the Transvaal Sub-group of the Ophthalmological Society of South Africa (M.A.S.A.) on 15 August 1960.

such cases. There should be proper equipment to investigate, treat, and record, so that the types and results could be classified.

2. A *glaucoma section*, where the different varieties could be thoroughly investigated, treated, and again recorded for research and teaching purposes, the equipment to include all methods of investigation, i.e. tonometers of all types, field charting apparatus, gonioscopes, etc.

3. A *uveitis clinic*. No one, as yet, has discovered the true aetiology of uveitis, and as there is considerable material in South Africa for research purposes, I should like to see such a clinic set up. It is certain that without facilities for investigation we shall never even have a chance of being the first to discover the cause.

4. A *genetic department*, where all hereditary conditions could be investigated and recorded. In order to investigate, a trained social worker would be necessary to trace the family connections of any such cases, and arrange for them to attend for examination and recording. This department should also be in touch with any similar organization in the General Hospital. Electroretinography could be included in this section.

5. A *squint department*, where all cases could be investigated and treated. Here an *orthoptic department* would be essential, and a close touch would be kept with the genetic department.

6. A *grafting department*, where all the necessary tissues could be kept in the bank and thorough research be carried out on methods of preserving. The tissues would be at the disposal of all departments.

7. A *traumatic department*, where all injured cases could be seen and dealt with at the earliest opportunity, this department to have its own theatre, which should be available 24 hours in the day.

8. A *general department*, where all cases not requiring the services of the special departments could be dealt with adequately and recorded. Refraction might be considered a separate part of this general department.

9. A *pathological department*, for all the necessary investigations. It might be more practical to maintain a close contact with the South African Institute for Medical Research, or a firm of clinical pathologists, one member of whom is especially interested in eye pathology.

10. A *photographic section*, with facilities for taking black and white, colour, and ciné pictures. This section should, of course, be in close touch with all the other sections.

11. *Theatres*. An eye hospital would require at least 4 theatres to fill the needs of all sections. There should also be a special theatre for the light coagulator.

12. A *general recording department*, where the records of all the cases could be maintained, and where, in addition, a pathological library could be kept, which would make it easy to turn up records of past cases. In this department a statistician would be invaluable. In addition to all these sections, there should be a lecture hall for the demonstration of cases and for teaching, and where ophthalmologists could meet generally to discuss their difficulties.

We are very proud of our present reference library, for which we are indebted to Dr. A. Jokl, but I should like to see this housed at the proposed eye hospital. It would be a good suggestion to ask all ophthalmologists to contribute to a fund for the purpose of subscribing to journals and books which are not already available in this library.

Those are my general ideas. St. John's Hospital, which was intended for Europeans and non-Europeans, may have had such ideas in view, but it has not developed along these lines, mainly, I should think, because of its great distance from Johannesburg and its situation in the non-European area. It is now functioning for non-Europeans entirely and, as we all know, the follow-up of non-European cases is, in most instances, impossible. I have envisaged the establishment of a European hospital, which would facilitate research as well as cater for the needs of Europeans.

It may be argued that an organization of this nature could never function amicably, since the present trend is individualistic, but I am of the opinion that medical practice is slowly but surely becoming State-controlled, and we must make provision for this by planning such a hospital.

It will also be doubted whether funds could ever be raised to establish such an institution, but I think it would be worth placing these ideas, or similar ones, before the proper authority for, if our case is stated well enough, we might get a sympathetic hearing. Once the Provincial authorities could be brought to our way of thinking — that is, that such a hospital would be of tremendous help to the community — financial assistance might be forthcoming, not only from the Province, but from employers of labour, such as the railways, and mines and industry in general.

In most cities in Europe there are eye hospitals functioning as units, and from these have emanated some of the world's foremost authorities on ophthalmology. Even so, these European institutions lack many features which could add to research and teaching, if the different branches were more coordinated. In effect, I feel that we could learn from their experience and perhaps start where they have left off.

In these troubled times, this may appear to be a pipe-dream, but I feel that its need to the community, both European and non-European, is so great, that, if this country continues to thrive, such a plan will become essential and will be a source of great satisfaction to the whole populace and to the medical profession in particular. You will, of course, appreciate that the ideas I have placed before you are only my very tenuous thoughts on this subject, and if we ever see a possibility of such a hospital materializing I know that among us we will have many more, different and better suggestions.

AMPTELIKE AANKONDIGING : OFFICIAL ANNOUNCEMENT

INSURANCE COMPANY MEDICAL AID

On 1 September 1960 a meeting was held between a Sub-Committee of the Executive Committee of Federal Council and representatives of SANSOM and the South African Mutual Medical Aid Society, in order to discuss certain difficulties which have arisen, including matters in connection with the approval by the Association of groups administered by them as medical aid societies and certain actions by the insurance companies which were prejudicial to the doctor-patient relationship. A report on the results of these discussions will be presented to the next Meeting of Federal Council.

Among other things, discussion took place in connection with certain cheques at present being issued by the South African Mutual Medical Aid Society to medical practitioners. On these cheques a certain code number indicates that the value of the cheque is according to the medical aid tariff of the Association and is '*tendered* in full settlement of the gross amount of such account'. The Sub-Committee stated its objections to this practice, but the representatives of the Society having explained the reasons for its introduction, stated that the Society was not in a position to discontinue it. Since important principles are involved the Sub-Committee decided that no final directive could be given to members of the Association at this stage other than to advise them to hold these cheques wherever possible and not to receipt or deposit them until after the next Meeting of Federal Council. As soon as possible after the meeting of Council, which is to be held on 19, 20 and 21 October 1960, the decision of Federal Council in this connection will be conveyed to members of the Association by means of a notice in the *Journal*.

A. H. Tonkin
Secretary

Medical House
Cape Town
8 September 1960

Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

DIE GESINSJAAR EN DIE GESINSKONGRES

Gedurende die afgelope tye het dit al meermale gebeur dat die aandag van welmenende mense dwarsoor die wêreld op spesifieke fasette van die algemene gemeenskapsprobleem gevestig is deur die instelling van spesiale en spesifieke propaganda-tydperke. So is daar, byvoorbeeld, die ‚Geestesgesondheidsjaar' wat gedurende 1960 deur die Wêreldfederasie vir Geestesgesondheid ingestel is om die aandag van so veel mense as moontlik te vestig op 'n positiewe benadering van probleme in hierdie verband.[1] So was daar ook die Internasionale Vlugtelingejaar[2] waarmee dit die doel was, nie net om die aandag op die moeilike probleme van die miljoene vlugtelinge wat daar orals oor die wêreld is te vestig nie, maar ook om pogings in werking te stel om vlugtelinge by te staan by die ingewikkelde en moeisame prosesse van heraanpassing en rehabilitasie.

En so het daar ook in ons eie land die ingewing ontstaan om 'n Gesinsjaar te beplan — 'n onderneming wat dit hom ten doel stel om te ywer vir die behoud van 'n gesonde gesinslewe en die bevordering van gesinseenheid. Dat die behoefte ontstaan het om so 'n Gesinsjaar te beplan, is goed te verstaan. Gedurende die afgelope aantal dekades het daar groot en verreikende veranderinge ontstaan in die basiese patroon van ons groepsverhoudinge.

Die ‚wind van verandering' wat so opvallend sterk oor die Vasteland van Afrika, sowel as oor die res van die wêreld, waai, is nie net 'n wind wat die hergroepering van politieke kragte aanblaas nie. Dit is 'n wind wat ook in ons land groot maatskaplike omwentelinge gebring het en wat die gemeenskap nie net in sy uiterlike aansig nie, maar ook in sy innerlike struktuur radikaal verander het.

Soos dit die geval is met die meeste volke van die Westerse wêreld het ons in 'n groot mate 'n bevolking van stadsbewoners en dorpsbewoners geword. Daarby het 'n ongesonde groot persentasie van ons mense woonstelbewoners en selfs kamerbewoners geword. Ook het die stryd om te bestaan hewiger geword en strominge soos materialisme, sekularisasie, individualisme, sensasielus, en geestesvervlakking het niemand onaangeraak gelaat nie.

Dit is dus nie te verwonder dat die hele basiese instelling van die gesin grootliks in die gedrang gekom het nie. Dit kom al meer voor dat altwee ouers deeltyds of voltyds buite die huis werk. Kinders word van vroeër af aan hulself en aan strominge buite die gesin oorgelaat. Die emansipasie van die jeug het vroeër 'n onverbiddelike finaliteit geword, en nuwe sedes en nuwe waardes het as meetsnoere en die gebruike van die vadere verdring.

Hierdie omstandighede het dus daartoe gelei dat alle organisasies wat met gesinsorg gemoeid is weer aan die probleme in verband met die huisgesin begin dink het, en dat verantwoordelike navraag en navorsing oor die hele probleem onderneem is. Die algemene opvattinge in hierdie verband, soos saamgevat in die brosjure waarin die betrokke organiserende komitee 'n Gesinsjaar vanaf die Uniefees in 1960 afgekondig het en 'n Gesinskongres in April 1961 aankondig, is soos volg:

1. Die deursnee huisgesin is nie toegerus en opgewasse om sy eenheid in 'n veranderde wêreld te handhaaf nie. Die huis het merendeels die uitgangspunt vir individuele lewe geword en is nie meer die middelpunt van saambinding nie.

2. Wantoestande soos konflikte, egskeiding, drankmisbruik, kinderverwaarlosing, jeugmisdaad, en ander, moet gesien word as simptome van 'n worteltoestand wat in sy kern ongesond is. Oorsake en nie simptome nie, moet behandel word.

3. Die probleemtoestand is baie kompleks met veelvuldige fasette. Al die geestelike, maatskaplike, funksionele en ekonomiese belange van die huisgesin is betrokke. Die oplossing kan nie deur middel van negatiewe veroordelings, vrome wense of oppervlakkige slagspreuke bereik word nie.

4. Slegs langs die weg van 'n positiewe benadering en 'n daadkragtige optrede, wat rekening hou met alle oorsaaklike invloede, sal 'n oplossing gevind word. In die lig van veranderde omstandighede moet daar 'n herontdekking wees van blywende gesinswaardes.

Die gedagte om so 'n Gesinsjaar in te stel, is kort voor sy aftrede as Administrateur van die Transvaal deur dr. Wm. Nicol uitgespreek, met die bedoeling dat daar gewerk sal word vir die behoud en die bestendiging van die gesinslewe. Hy het aan die hand gedoen dat die Gesinsjaar uit die Uniefees moet voortvloei en dat almal die geleentheid sal hê om 'n positiewe bydrae te lewer tot herstel van gesinseenheid en die bevestiging van wesentlike gesinswaardes.

Hierdie gedagte het onmiddellik 'n gunstige weerklank gevind en dit het uitgeloop op die samestelling van 'n sentrale reëlingskomitee en die beplanning van 'n omvattende kongres wat in April 1961 in Pretoria gehou sal word, waar alle aspekte van die gesinslewe onder die soeklig geplaas sal word. Langs hierdie weg en namens die mediese professie in Suid-Afrika, wil ons graag ons ondubbelsinnige ondersteuning aan hierdie lofwaardige voorneme toesê in die hoop en verwagting dat ook hierdie poging sal bydra tot die verwesenliking van die ideaal van die skepping van 'n gesonder, gelukkiger, en meer stabiele wêreld in die toekoms.

1. Van die Redaksie (1960): S. Afr. T. Geneesk., **34**, 175.
2. *Idem* (1960): *Ibid.*, **34**, 685.

ATRIAL SEPTAL DEFECTS

Atrial septal defect is one of the common types of congenital heart disease. Although this condition was recognized for centuries and received increased attention since 1900, the first authoritative clinical account was provided by Bedford, Papp, and Parkinson in 1941, who described the physical, electrocardiographic, radiological,

and diagnostic features, as well as the complications and clinical course. With the advent of new diagnostic techniques, many publications have described the pathological physiology of the disease. Finally, the introduction of surgical correction has given impetus to further inquiries into the many facets of this abnormality.

During foetal life, the left ventricle receives almost no blood from the lungs, and must therefore be supplied by the venae cavae and the common atrium if it is to perform any work to help in its proper development. Herein lies the reason for the apparent complexity in the development of the interatrial septum, in which the septum primum, septum secundum, and endocardial cushions all play an important rôle. The septum is so built that it will at all times in foetal life allow blood to flow from right to left, but at birth this one-way valvular partition must be readily closed by the rise of left atrial pressure.

From the practical surgical angle, three chief varieties of atrial septal defects are recognized. Their pre-operative differentiation is not only possible but also desirable, since the surgical correction for each type varies.

1. *Sinus venosus defects.* These defects occur at the root of the superior vena cava above the fossa ovalis. They are commonly associated with anomalous drainage of the right superior pulmonary vein.

2. *Ostium secundum defects.* Secundum defects are more centrally placed in the septum. The margins of a secundum defect are formed by remnants of the atrial septum and thus the tricuspid valve is separated from the defect by a rim of septal tissue. The opening of the inferior vena cava may be partly or wholly on the left atrial side of the inferior rim of the defect.

3. *Ostium primum defects.* These abnormalities are also known by terms such as atrioventricular defect or common atrioventricular canal, but 'endocardial-cushion defect' would appear to be the best name, since there is always evidence of failure of the endocardial cushions to develop normally and fuse in the midline of the common atrioventricular canal. In addition to defective development of the atrial septum, there are thus abnormalities of varying degree in the leaflet formation of the atrioventricular valve and of ventricular septation.

Various ingenious closed or external techniques of repairing the defect were devised by Cohen, Gordon Murray, Bailey, Sondergaard, and Edwards. Gross, in 1952, advocated a semi-open technique of suturing a rubber well to an incision in the right atrium, thus allowing direct suture by tactile control.

The advent and perfection of hypothermia, however, provided a more direct approach. Closure by this method was first carried out by Lewis and Taufic in 1953, and subsequently extensively practised all over the world.

As experience and confidence in pump oxygenators increased, this technique gradually superseded general hypothermia, because the operative risk of total cardiopulmonary bypass is as low as general hypothermia and, using this technique, more time is available for careful suture of the defect, with the use of a plastic patch in the larger and more complicated defects. There is no doubt that in the repair of endocardial-cushion defects, only open cardiotomy with extracorporeal circulation should be used.

In selecting patients with uncomplicated atrial septal defects for surgery, it must be remembered that the prognosis in these cases is very good; without surgical treatment most of these patients will reach middle age without any disability. Before surgical correction is undertaken, the patient must, therefore, be guaranteed a complete cure with an operative risk approaching zero per cent.

On pages 797, 805, and 810 of this issue of the *Journal* we publish three articles by various authors in which both the medical and surgical approaches to various facets of the problem of the treatment of atrial septal defects are discussed and evaluated.

TAALRUBRIEK

Die Taalkomitee van die Geneeskundige Skool van die Universiteit van Stellenbosch stel voor om te gebruik:

1. Eng. dumping: Afr. *dumping* (en bv. *dumpsindroom*). Spreek uit met *u* soos in *tulp*, dus nie ‚damp' nie.
2. Eng. scalp: Afr. *kopvel* (s. nw.). Waar nodig, sou ons dus kon praat van die *huid* van die kopvel.
3. Eng. fissure: Afr. *fissura*, -s, en in sekere verband ook bv. *spleet*, *skeur*.
4. Eng. menarche: Afr. *menarg*.
5. Eng. Mendelian recessive: Afr. *Mendel se resessief* of (die) *Mendel-resessief*; vergelyk ook Naboth se follikels, Fallopius se buis, en ander.
6. Eng. tubule: Afr. *buis* of *buisie*, en in spesifieke gevalle word dan bv. gepraat van Eng. tubular necrosis: Afr. *nierbuis-nekrose*, e.d.m.
7. Eng. plication: Afr. *plooiing* of *plisering*.
8. Eng. transmitted pulsations: Afr. *oordragingspulsasies* of *oorgedraagde pulsasies*, waarby dan ook: *pulsasies word oorgedra*.
   Eng. expansile pulsations: Afr. *uitsettingspulsasies*.
9. Eng. condyle: Afr. *kondiel*, mv. *kondiele*.
10. Eng. Physical Medicine: Afr. *Fisiese Geneeskunde*, teenoor *Geneeskundige Fisika* (fisikus), en naas *Fisioterapie* as vak en as behandelingsvorm.

Korrespondensie

Die Komitee kry baie briewe met waardering en probleme. Dit is goed. Maar die lesers help nie genoeg nie. Nou vra die Komitee weer raad: help asb. met Eng. *crush injury*.

OPEN-HEART SURGERY IN ATRIAL SEPTAL DEFECT*

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Atrial septal defects are probably the commonest of the congenital heart lesions, and as the name indicates they consist of some form of abnormal communication between the two atria. The defect may occur as a hole in the interatrial septum itself or, more complexly, as a hole in the lower part of the interatrial septum where it merges into the ventricular septum. This latter may be due to an additional defect of the ventricular septum, and of the leaflets of the tricuspid and mitral valves, which are derived from the endocardial cushions.

An atrial septal defect may therefore be regarded as being interatrial or atrioventricular (an endocardial cushion defect). The different terminology that is applied to these two types, according to embryological descriptions, tends to make the subject unnecessarily difficult to understand.

The defect in the atrial septum may be large or small, single or multiple. Occasionally, it may resemble a cribriform plate instead of a simple hole. If the defect is very large the appearance may be that of a single atrial cavity, the only sign of the septum being a faint rim on the atrial wall.

It is important to remember that an abnormality of the pulmonary veins may coexist with the developmental atrial defect. The drainage of these veins may be anomalous, i.e. they may drain into the right atrium instead of the left; or may appear to be anomalous in the case of a very large atrial septal defect, the impression being that the pulmonary veins are opening into the right atrial cavity. Occasionally the right superior pulmonary vein drains directly into the superior vena cava.

The presence of an atrial septal defect is of no importance during intra-uterine life, because the normal course of all the venous blood then entering the heart is to the left side *via* the fossa ovalis.

The left ventricle shows greater development than the right during the first year of the child's life because of the greater resistance encountered in the peripheral circulation. Consequently, many cases of atrial septal defect remain unrecognized for this length of time. Because of the defect, the blood flows from the left into the right atrium, for there is normally a higher blood pressure in the left chamber. The right atrium, which now has to accommodate all the blood it receives from the two venae cavae, as well as this extra blood from the left atrium, enlarges correspondingly. The right ventricle receives an increased flow from the right atrium, and accordingly its action becomes more forceful. The additional blood volume is pumped into the pulmonary arteries, which enlarge and show pulsation as a result of the extra force transmitted from the right ventricle. This explains the left-to-right shunt with increased strain on the right ventricle. The resultant additional flow into the pulmonary arteries is responsible for the systolic murmur.

The enormously increased strain on the right ventricle is often well tolerated, but eventually failure occurs. This event, which takes 20 - 30 years to develop, is of serious prognostic significance. Of course, if the response to the right-sided over-activity is pulmonary hypertension, reversal of the shunt may take place. A balance may be secured for a time but finally a right-to-left shunt develops with cyanosis, which is an ominous portent.

History of Treatment

Recognition of these cases has become a routine matter within the last 2 decades, and today the diagnosis can be made clinically with great accuracy. Great advances have been made in methods of treatment in the last 10 years. The following résumé of the work that has led to present-day techniques should prove of interest:

In 1947 the closure of an experimental defect by Cohn, by suturing the atrial wall to the margin of the defect, was the forerunner of Murray's successful closure of a defect in a child in 1948. In 1950 Swan attempted to plug the hole by the inversion of the auricular appendage. Bailey devised the doughnut-lumen type of atrioseptopexy in 1952, and a similar method was employed by Hufnagel and Gillespie in 1951, when they used a plastic button to close the opening. Sondergaard described a technique of defect-closure by insertion of an external circumferential suture, and Gross showed that it was possible to work in the left atrium through a rubber well attached to the atrial wall.

Many patients were treated by these methods, but it was obvious that a more accurate means of closure was necessary because of the great variability in size, number and position of these defects. In 1953 Lewis and Taufic were able to visualize and close defects under direct vision, utilizing hypothermia and inflow stasis of blood. This method allowed them about 7 minutes in which to operate, during which time all blood flow to the heart was cut off by ligatures around both venae cavae and a clamp across the aorta and main pulmonary artery. The wall of the right atrium was opened, and the defect was usually accurately and completely repaired. Apart from the time-limit set the surgeon, the disadvantage, of course, was that during this time all cerebral circulation ceased. Simple defects, even if large, could be closed by this procedure with absolute certainty. More complicated cases, however, with venous anomalies or with endocardial cushion defects, could not be treated in the short time at the surgeon's disposal.

Extracorporeal circulation now gives the surgeon ample time to correct all types of defect.

CASE INVESTIGATION

Clinical Signs

Many cases are known to have had heart murmurs for several years. These may have been discovered in a routine examination, or an examination for some unrelated complaint. Schoolchildren may show some degree of dyspnoea, or complain that they are unable to keep up with the activity of their schoolmates. In many instances, however, the symptoms are minimal. Some children may show minor degrees of physical retardation, while others are normally developed, the heart murmur being the only sign of any

* Paper presented at the 42nd South African Medical Congress (M.A.S.A.), East London, C.P., September-October 1959.

disability. The pulse may be normal, and this is always a striking finding in the presence of cardiac signs. A precordial chest bulge may be present and a systolic thrill may be palpable. A systolic murmur with a widely-split second pulmonary sound is audible over the third left intercostal space.

A diastolic murmur may be heard if pulmonary insufficiency is present. Frequently a diastolic tricuspid murmur is present as a result of the increased flow through the valve. A diastolic mitral murmur suggests an atrioventricular defect (AV type of defect).

Females are affected twice as often as males.

There is usually no history of maternal ill-health during the pregnancy, and the mother may have had normal children before and after the birth of the affected child.

X-ray Screening

This examination is of the utmost value. The heart is enlarged and hyperactive, and the hyperactivity extends to the pulmonary arteries, which may show increased activity into relatively small branches. In contrast, the left atrium, left ventricle and aorta show lack of prominence. This finding has been named 'hilar dance'.

Cardiac Catheterization

Important information is gleaned from this examination, although the diagnosis of an atrial septal defect can usually be made without it. In many cases, however, an AV-canal type of defect, as distinct from the ordinary intra-atrial type, can only be diagnosed after catheterization.

An anomalous venous drainage may be demonstrated by passing the catheter directly from the superior vena cava into the superior pulmonary vein. The position of this catheter will be seen on the straight X-ray film, and the nature of the defect is confirmed by the high oxygen saturation of the blood samples withdrawn through the catheter. A left-sided superior vena cava may be shown on the AP view in an X-ray film. This finding should be confirmed by lateral views, for the catheter may have been passed down the left internal mammary vein giving the appearance of a left superior vena cava.

The passage of the catheter from right to left will confirm the presence of a communication between the two atria. The catheter will pass at a higher level in a case of ordinary intra-atrial defect than in one with an AV type of defect. The passage of the catheter directly from the left atrium into the left ventricle, pressures, position of catheters, and oxygen saturations, all provide confirmatory evidence of the nature and position of the defect.

It is important to remember that though catheterization often demonstrates the anomalous pulmonary veins, the absence of this finding does not preclude their existence.

The surgeon is thus usually provided with an accurate pre-operative picture on which he can base his plan of action.

Electrocardiograph

ECG tracings usually demonstrate partial or complete right bundle-branch block, or right ventricular hypertrophy. Signs of left ventricular hypertrophy or complete heart block are suggestive of an atrioventricular type of defect.

OPERATION FOR ATRIAL SEPTAL DEFECT

The operation is performed under *cardiopulmonary bypass*.

The anaesthesia is a combination of pentothal, relaxant, gas, oxygen, and fluothane, and should be sufficiently light to allow the patient to become wakeful immediately after the operation.

The patient is placed in a supine position with a narrow pad between the shoulders, and a mid-line sternum-splitting incision is made. The sternum is divided by a Gigli wire saw or an electric motor saw, and the pleural cavities are not opened. Alternatively, a right posterolateral incision through the one pleural cavity or a bilateral transthoracic incision through both pleural cavities can be used.

Arterial and venous pressures are monitored through polyethylene tubing in the internal mammary artery and the long saphenous vein. Electrodes in the scalp and on the extremities provide leads to the EEG and ECG machines respectively.

Arterial cannulae of suitable size are prepared, and the pump oxygenator is calibrated according to the patient's weight.

The heart is exposed by a long incision in the pericardium so that good access is obtained to both venae cavae. Appropriate venous catheters are prepared.

Preliminary investigations may have suggested the presence of anomalous veins, but one must remember that the absence of any pre-operative evidence does not exclude their presence.

A persistent left-sided vena cava allows uncontrollable amounts of blood to enter the right atrium during bypass if it is not clamped off. It is thus imperative to examine the left side of the heart as well as the posterior aspect, for if there is such a vein it will enter the coronary sinus.

An aberrant superior pulmonary vein may be seen entering the superior vena cava. Occasionally aberrant pulmonary veins may be seen entering the right atrium, though this abnormality is usually only confirmed when the right atrium has been opened.

The following principles are worth remembering:

1. Anomalous pulmonary veins must have their blood flow diverted to the left atrium.

2. The blood flow from the venae cavae must be diverted to the right atrium. This applies particularly to the inferior vena cava when there is a low-lying defect and a well-developed eustachian valve, which is sometimes mistaken for part of the rim of the defect. During inflow stasis under hypothermia the valve may be caught up and used to repair the defect, so that inferior-vena-caval blood is diverted into the left atrium. This error, of course, is obviated by the use of the cardiopulmonary bypass.

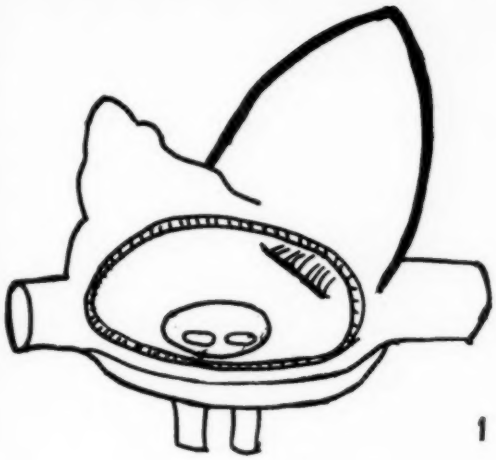
3. A preliminary digital exploration of the defect through the right atrial appendage is always essential. The surgeon may not only establish the presence of mitral stenosis but may also be able simultaneously to perform a digital valvotomy.

4. A piece of prepared ivalon sponge should be ready in case it is required for the closure of a badly situated defect. This applies particularly in the case of an atrioventricular abnormality.

Repair of Interatrial Defects

Interatrial defects can be classified into 3 groups, as follows:

1. *The high or sinus-venosus type* is a defect situated at the lower end of the superior vena cava. This type of defect will require an ivalon patch if the defect extends up into



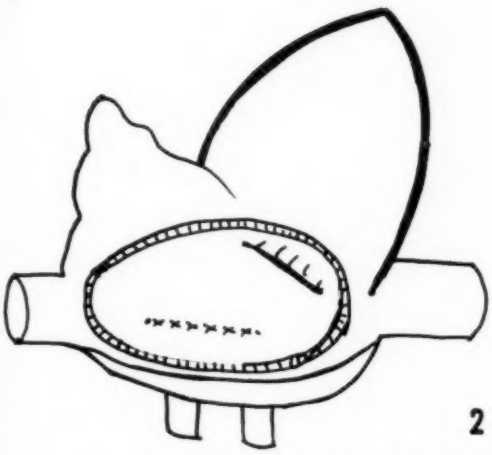


Fig. 1. This illustrates the presence of a septal defect situated in the middle zone of the septum. Through this defect can be seen the opening of the 2 left pulmonary veins, and they open onto the left side of the septum.

Fig. 2. The septal defect has been converted into a horizontal slit by the insertion of a layer of sutures, and when this is over-stitched, the septum is completely closed off.

the superior vena cava. Mattress sutures must be inserted through the wall of the superior vena cava from the outside at the top part of the defect. There is usually a rim of atrial septum in the lower part and the mattress sutures can be inserted from the left atrial side. When the defect has been circumferentially supplied with mattress sutures, the ivalon patch can be slid down into position and fixed. This defect may be associated with an anomalous pulmonary vein, in which case the veins must be diverted before the repair of the defect. Small anomalous veins may be left alone.

2. *The middle type* of defect is probably the commonest variety (Figs. 1 and 2). The defect may be very large, and there is always a rim of atrial septum. This round opening can be converted into a slit-like opening by placing a mattress suture at each end and applying slight traction to each suture. The slit-like opening is then stitched upwards and downwards with a continuous suture, care being taken that a firm hold is obtained with each stitch through the septum. Other openings are treated similarly. An extra opening may be so small that a single mattress suture is sufficient to close it.

3. *The lower type* of defect extends down to the region of the eustachian valve of the inferior vena cava, and there may be no visible rim of atrial wall. An artificial rim can be made by picking up the atrial wall in sutures, which will then help to convert the rounded defect into a slit-like opening. The presence of the caval catheter will be of great help, for it will ensure that the flow from the inferior vena cava is not diverted into the wrong atrium. This is a point in favour of the use of the cardiopulmonary bypass as opposed to hypothermia.

The septal defect in any one of these 3 types may be so large that it is impossible to suture the margins together without causing great tension on the stitches. The stitches are thus likely to tear out and lead to a recurrence of the defect and it is therefore deemed wiser to stitch an ivalon patch over the defect. A piece of previously prepared ivalon is cut into the correct shape and attached to the margins of the defect by means of mattress sutures.

Care must be taken to allow all air to escape from the left atrium before the final stitch is drawn taut; this applies to direct suture as well as to a patch. During the repair operation the suction apparatus must be used to ensure visibility of the site. Sometimes there is quite a large blood flow into the left atrium, either from bronchial arteries which return their blood to the pulmonary veins, or most commonly from the coronary sinus *via* the right atrium. When the blood is removed from the operation site the left atrium may be emptied, and any air which enters may

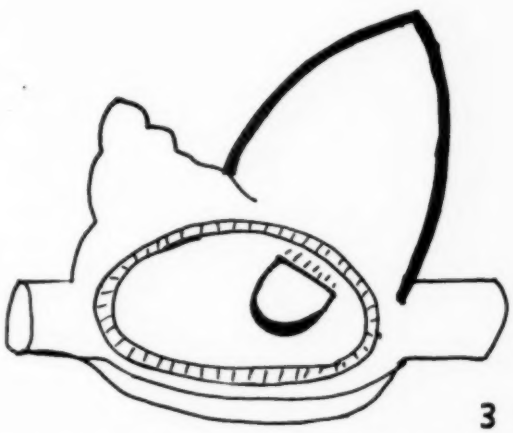


Fig. 3. This illustrates an ostium primum septal defect situated immediately over the tricuspid valve. The tricuspid and mitral valves may be split, in which case the defect would be of atrioventricular-canal type.

pass into the left ventricle. This is extremely dangerous, for this air may be pumped by the ventricle into the aorta, from where it may filter into a coronary artery, or into the cerebral or systemic circulation.

The Atrioventricular Type of Defect

A low defect, with part of its inferior margin formed by the septal leaflets of the mitral and tricuspid valves, is called the atrioventricular type (Fig. 3). These two valves are rarely intact, and their repair must be performed before the closure of the atrioventricular defect. It is usually possible to repair the clefts by simple suturing, but at times it may be necessary to use a narrow strip of ivalon, which can be sewn to both sides of the cleft. It is important to mobilize the two cleft leaflets very thoroughly before repairing them. After repair of the valves the defect in the atrial wall can then be closed by means of an ivalon patch.

Frequently not only are the two valves divided, but there is a failure of development of the upper part of the interventricular septum. This means that there is an interventricular communication as well as an interatrial communication. The valves developed from the cushions of the septum primum are also divided, so that there is an additional communication between the left ventricle and the right atrium. This abnormality can be clearly shown on catheterization, when the catheter can be seen passing from the right atrium directly into the left ventricle through a low-sited defect.

It is important to ascertain that there is no air trapped in the left ventricle when the mitral valve has been repaired. An incompetent mitral valve cannot force the entrapped air in the ventricle into the aorta, but a competent mitral valve after repair will offer such resistance to the entrapped air that it may be forced up into the aorta. This complication is readily recognized on the ECG tracings. The arterial blood enters the patient from the pump oxygenator at the right femoral artery, and the stream of blood is therefore against the normal direction of blood in the aorta. Consider what happens when blood and some air is ejected from the left ventricle through the aortic valves and into the aorta, which is receiving blood in an opposite direction. The mixture will tend to go down the coronaries, and the air in these vessels will show an immediate pattern of damage on the ECG. This accident can be treated by keeping the patient on the pump so that eventually all the air will be driven out of the coronaries, with the consequent restoration of a normal ECG pattern. It is far more serious if air gains entrance to the cerebral vessels. The mitral valve should therefore be rendered incompetent temporarily until the surgeon is reasonably sure that all air has left the ventricle.

The existence of this type of defect gives further support to the theory that heart valves develop from the endocardial cushions. These two cushions develop ventrally, and dorsally, and they then fuse. If there is failure to fuse, then either the left-side valve, i.e. the aortic leaflet of the mitral valve, or the right-side valve, i.e. the septal leaflet of the tricuspid, will remain cleft. In many instances, both remain cleft.

The septum primum grows down to join with these fused cushions, dividing the space into the two atria. Directly beneath the two atrioventricular cushions is the muscular part of the ventricular septum. The interventricular opening is obliterated when these cushions fuse to form part of the membranous septum between the two ventricles.

The malformation may be complete, that is, it may involve both mitral and tricuspid valves, or partial, involving only the mitral valve. The complete form shows interventricular communication as well as insufficiency of both mitral and tricuspid valves. The partial form may show insufficiency of the mitral valve.

Modern treatment of these defects of atrioventricular type is carried out under direct vision and can be performed in two ways, viz. (1) under hypothermia with inflow stasis or (2) under cardiopulmonary bypass.

Hypothermia allows the surgeon a limited time in which to do the repair. The atrium is opened and the defect is visualized and then repaired. Occasionally more time is required, and it may be necessary to have two periods of inflow stasis. Obviously this method can only be employed for the simpler types of interatrial defects. Repair of the more complicated types, particularly the AV-canal type of defect, requires considerably more time, and *cardiopulmonary bypass* becomes essential.

Anomalous Venous Drainage

Anomalous pulmonary veins drain either into the superior vena cava or into the right atrium. The presence of these anomalous veins with an interatrial septal defect increases the difficulty of repair. All pulmonary venous drainage should pass into the left atrium, and closure of an interatrial defect without correction of the pulmonary venous drainage would result in a continuation of the left-to-right shunt; that is to say, the original state would still be present, though to a lesser degree.

The operation for the correction of the anomalous venous drainage must be so devised that the flow from the anomalous veins is directed into the correct atrium, i.e. the left atrium. In the case where the superior pulmonary vein drains into the superior vena cava, the aim of operation is to separate the oxygenated blood in the pulmonary vein from the de-oxygenated blood in the superior vena cava. A row of mattress sutures can be inserted into the superior vena cava, dividing it into two streams. The stream from the pulmonary vein can then be diverted into the left atrium by bringing a flap of atrial wall over the newly-made venous orifice. This flap is formed by making a curved incision in the atrial septum at the new opening of the pulmonary vein, and stitching this flap to the atrial wall. The atrial septal defect is closed in the usual way.

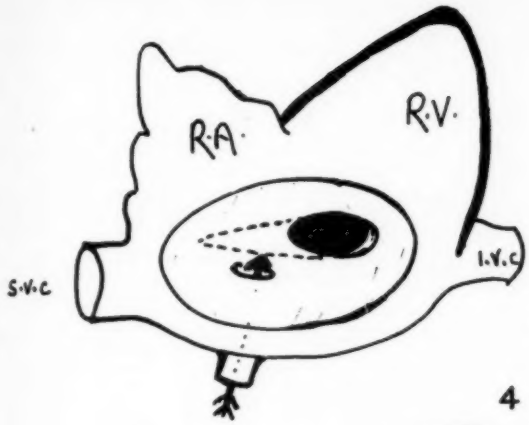
In the case where a pulmonary vein drains into the right atrium, a flap is fashioned from the atrial wall adjacent to the septal defect, and this flap is then stitched to the atrial wall (Figs. 4, 5 and 6).

In both cases, if the newly fashioned septal flap is not large enough to be attached to the atrial wall, a section of compressed ivalon can be used as a patch.

The purpose is to ensure that all oxygenated blood is diverted to the left atrium, and that the septal defect is closed.

Completion of Bypass Operation

The use of cardiopulmonary bypass allows the surgeon ample time to examine the defect thoroughly and to close it either by direct suture or by means of ivalon patches. Adequate perfusion and blood oxygenation can be carried out



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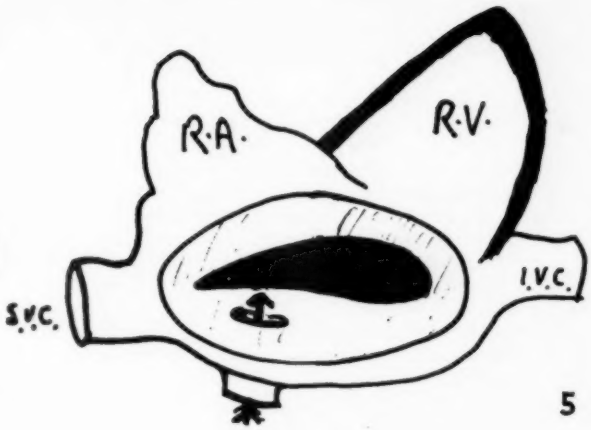

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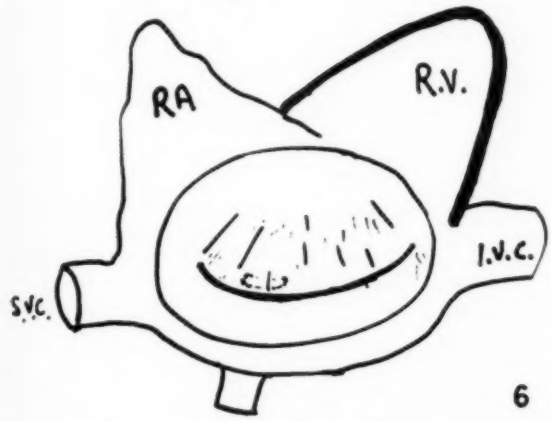

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Fig. 4. This shows an oval-shaped defect with an anomalous pulmonary vein opening onto the right side of the septum. The correction of this defect is started by enlarging it along the dotted line.

Fig. 5. The defect has now been enlarged considerably and the upper margin has to be brought over the opening of the pulmonary vein so as to divert its flow into the left atrium.

Fig. 6. This shows the completed repair with the anomalous vein now draining into the correct atrium.

with any of the different types of pump oxygenator. The Cape Town unit at Groote Schuur Hospital uses the bubble oxygenator.

After closure of the defect, the right atrium is allowed to fill with blood while the atriotomy incision is repaired. Haemostasis is all-important, and a careful closure is essential.

When preparations are made to go off bypass, the superior-vena-caval snare is released and then the inferior. The effect on the heart beat is noted, and particularly the appearance of the ECG pattern. If all is well, the machine is stopped and the arterial and venous lines are clamped. The superior-vena-caval catheter is removed and the previously applied purse-string suture is drawn taut and then tied off. The inferior-vena-caval catheter is similarly removed and an extra purse-string suture is usually inserted because this catheter is placed in the wall of the atrium. The superior-vena-caval catheter is placed through the appendage of the atrium.

When all the incisions have been closed off and inspected, the internal-mammary cannula is removed and the vessel is tied off. The chest is then ready for the placing of drainage tubes. The anterior mediastinum should be drained by 2 catheters and, if the pleural cavity has been opened, a drainage tube should be inserted into the cavity.

Dressings are applied, the patient is weighed to check blood loss or gain, and an X-ray film is taken of the chest to confirm that the lungs are fully expanded and the drainage tubes correctly placed.

Postoperative care is not to be discussed in this paper, but it should be stated that these patients remain under constant supervision by senior medical and nursing staff for the first 48 hours of the postoperative period.

ANALYSIS OF CASES

The technique of cardiopulmonary bypass has been used at Cape Town for 52 cases, 20 of which were suffering from atrial septal defects. Of the 20 cases, 16 were in the group of interatrial septal defects, i.e. the 'ostium secundum' group, and 4 were in the 'ostium primum' or endocardial-cushion group. There was 1 death, which occurred in a case of the 'ostium primum' type. All cases catheterized since operation, so far, show complete closure of the defects.

CONCLUSIONS AND SUMMARY

This report deals with the surgical treatment of 20 cases of atrial septal defect. A panorama of treatment is given. The closed methods should not be used today, although the open methods under hypothermia with a limited operating time are still used by some surgeons. Here I have discussed the precise surgical technique which is possible with cardiopulmonary bypass.

The technique which we prefer and is in use at present, viz. cardiopulmonary bypass, allows us to repair completely all types of defect with minimum risk, and without the strain on the surgeon of an imposed time limit.

ATRIAL SEPTAL DEFECT*

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Atrial septal defect constitutes the commonest of all congenital heart anomalies. In most cases, however, there are associated cardiac anomalies[1] and frequently the defect in the atrial septum is of secondary importance. Isolated atrial septal defect is nevertheless still a common defect and, in our experience in Johannesburg, it occurs as frequently as isolated ventricular septal defect, these two lesions being less common, however, than patent ductus arteriosus.

We present here our experience in 43 patients, 37 with isolated atrial septal defect and 6 with persistent common atrioventricular canal.

Material

The patients were all seen personally by one or more of the authors. The diagnosis was supported by cardiac catheterization in 28 cases. In 17 of these 28 the diagnosis was confirmed at operation for closure of the defect, and in 3 of the 17 necropsy examination added further confirmation. In 7 more cases the diagnosis was confirmed at necropsy. In the remaining 8 cases the diagnosis was made with confidence on clinical grounds and cardiac catheterization was advised.†

Pathological Anatomy

In recent years much confusion has arisen as a result of different anatomical classifications of atrial septal defects. Without entering into any controversy regarding the embryology of the defects, we have attempted to classify our cases into the following 2 groups only. We believe that the mode of surgery should depend on which of the 2 types of defect is present:

1. '*Septum secundum*' *defects*. The defect is usually in the situation of a foramen ovale, but may be higher ('sinus venosus' defect) or lower in the septum. The ventricular septum is intact and the atrioventricular valves have no congenital abnormality. Surgery under hypothermia is the method of choice.

2. '*Septum primum*' *defects*. The defect is always low, and there may be associated congenital anomalies of the mitral valve and/or the tricuspid valve, and possibly a defect of part of the ventricular septum (common atrioventricular canal). Extracorporeal circulation is essential for surgical repair.

While our classification may not satisfy embryologists, we have found it to be satisfactory for clinical application.

In our series, 37 were secundum defects, and 6 (14%) were primum defects.

Lutembacher's syndrome. By definition, this condition is an association of mitral stenosis and a sizable congenital interatrial septal defect. It is now accepted as being rare, and we had no case of it in our series. However, one of our patients had severe rheumatic mitral incompetence with a large atrial septal defect.

* Paper presented at the 42nd South African Medical Congress (M.A.S.A.), East London, C.P., September-October 1959.

† Two of these 8 patients have been catheterized since this paper was presented and the diagnosis has been confirmed.

Age and Sex Incidence

There were 30 female patients and 13 male (2·3 : 1). The higher incidence in females is well known.

The ages ranged from 3 months to 54 years (Table I). As in most series, the greatest number of cases occurred between 2 and 40 years of age. The fairly high incidence in the 1st year of life is due to the inclusion of 4 patients who were seen for a congenital heart anomaly in infancy, and who all died, the diagnosis being confirmed by necropsy.[2]

TABLE I. AGE INCIDENCE

Age (years)	*Patients*	%
0– 1	4	9·3
1– 2	2	4·6
2–10	12	28·0
10–20	12	28·0
20–30	5	11·6
30–40	6	13·9
40–50	1	2·3
Over 50	1	2·3

Prognosis

It will not be possible to arrive at an accurate assessment of the prognosis in atrial septal defect unless long follow-up studies of large numbers of patients are made. However, 2 facts become evident from analysis of our material: (1) When the patient with an atrial septal defect presents *clinical* evidence of heart disease in early infancy, the prognosis is grave. All 4 of our cases died within the first 2 years of life. We believe, although we have no definite proof, that these cases may be associated with a persistence of 'foetal pulmonary hypertension'. Histological examination of the lung vasculature at necropsy showed thickened media of the smaller arteries in 1 case. In the other 3 the pathologist regarded the vessels as normal. None showed intimal hyperplasia. Our experience would indicate that in those patients with a good prognosis in early life, *clinical* evidence of heart disease is generally not present in infancy. However, in one of our patients, aged 4 years, who was first seen at 6 months of age, although the heart was then regarded clinically as normal, an electrocardiogram showed an rsR′ pattern in VI, and this pattern persisted. (2) Atrial septal defect is rare over the age of 40 years. One of our patients, aged 44, had severe exertional dyspnoea but after corrective surgery her exercise tolerance became normal. Our oldest patient died at 54 of a cerebral thrombosis (? paradoxical embolus) following hysterectomy, and necropsy revealed a small atrial septal defect (0·5 cm. in diameter). Nevertheless there was considerable right ventricular enlargement and the patient had experienced severe dyspnoea.

Of the 6 patients in the 4th decade, 2 were in severe, intractable congestive cardiac failure and died, 1 after corrective surgery.

We consider, therefore, on the available evidence, that atrial septal defect is a serious congenital anomaly with a high mortality in early infancy and early middle age. Campbell *et al.*[3] reported that after infancy has passed the prog-

nosis remains good until the 4th and 5th decades. Similar figures are presented by Bedford *et al.*[4]

The following are factors which appear to affect the prognosis in the present series:

1. *Type of defect.* Of the 11 patients who died, 2 were suffering from primum defects; their ages at death were 2 years and 10 years.*

2. *Pulmonary hypertension.* Of the 9 deaths in patients with secundum defects, 6 probably had well-marked pulmonary hypertension. Of the 6, 4 were under 2 years of age and the other 2 were 22 and 23 years old. The 3 deaths in patients with secundum defects, but no significant pulmonary hypertension, were in patients aged 37, 39 and 54. We believe, therefore, that the prognosis is worse in those with primum defects and in those with significant pulmonary hypertension (Table II).

TABLE II. DEATHS IN RELATION TO AGE, TYPE OF DEFECT AND PULMONARY HYPERTENSION

Age (years)	*Primum defect*	*Secundum defect with pulmonary hypertension*	*Secundum defect without significant pulmonary hypertension*
0- 2	1	4	0
2-10	1	0	0
10-30	0	2	0
Over 30	0	0	3
Total deaths ..	2	6	3

CLINICAL PICTURE

The clinical picture of atrial septal defect in infants differs considerably from that in older children and adults; it will not be discussed here because it is the subject of a separate report.[2]

In children and adults, the patients are often of gracile habitus. Arachnodactyly and high arching of the palate are sometimes, though rarely, present; they were noted in only 3 of our cases. Mongolism was not present in any of our cases. Cyanosis and clubbing of the fingers were present in 1 patient, who had severe pulmonary hypertension with reversal of shunt.

The pulse is commonly of small volume. Venous pulsation in the neck is not a noteworthy feature, except in patients with congestive cardiac failure.

In patients with secundum defects there is usually prominent pulsation to the left of the sternum and in the 2nd left intercostal space. A systolic thrill is not often present; it was felt at the pulmonary area in 4 of our patients with secundum defects (10·8%). On auscultation of the heart, the following are classical features in these cases:

1. An ejection systolic murmur, usually of moderate intensity (grade 3 or 4), occasionally soft, and occasionally very loud, is best heard at the pulmonary area and, if loud, may be conducted all over the precordium.

2. The second heart sound is well split (0·03 - 0·06 second, but in 2 patients 0·02 second) and the degree of splitting varies little, if at all, with respiration. The 2nd (pulmonic) component of the 2nd heart sound is usually only slightly louder than the 1st component, but in patients with pulmonary hypertension it is markedly increased in intensity.

3. A systolic ejection click is commonly present; it was found in 23 (62%) of our patients with secundum defects. In some cases the click is difficult to separate clinically from the systolic murmur, but gives the murmur a 'coarseness' of quality. In these cases, the click can be demonstrated with the phonocardiograph, and it often becomes easily audible after surgical closure of the atrial septal defect, when the murmur frequently disappears.

4. A soft, short diastolic murmur, sometimes 'scratchy' in quality and occurring shortly after the 2nd heart sound, is frequently noted at the lower end of the sternum;[6] it was found in 26 (70%) of our cases of secundum defect. This murmur is believed to be due to 'torrential' flow across the tricuspid valve, and is usually best heard during inspiration.

5. A soft blowing early diastolic murmur, due to pulmonic incompetence, may be heard occasionally, especially in patients with pulmonary hypertension.

In patients with primum defects, the above features may all be present. In addition, a localized left ventricular heave may be felt over the cardiac apex. In all our 6 cases, a well-marked pan-systolic murmur could be heard at the apex, and in 4 of them this murmur could be distinguished from the pulmonary ejection murmur. In 1 patient, a loud pan-systolic murmur with associated thrill was best noted along the left sternal edge. In 5 patients, a well-marked mid-diastolic murmur was audible along both edges of the sternum and in 3 of them this murmur was well heard at the cardiac apex.

Symptoms referable to the heart were frequently present, at all ages, but were severer in the older patients and in those with primum defects or pulmonary hypertension. The degree of disability could not be well correlated with the degree of left-to-right shunt. Of the 6 patients with primum defects, 3 were in congestive cardiac failure with its attendant symptoms. Of the remaining 3, 1, aged 3½ years, was asymptomatic and 2, aged 12 and 33, experienced severe exertional dyspnoea and tiredness. Excluding the 4 infants who died, there were 33 patients with secundum defects, of whom 4 were in congestive cardiac failure. Of these 4, 2 were over 30 years of age; 1, aged 11, had associated rheumatic mitral incompetence; and 1, aged 2½, had moderate pulmonary hypertension. Dyspnoea and tiredness on exertion was present in 21 of the remaining 29 patients with secundum

TABLE III. PATIENTS ACCORDING TO SYMPTOMS

Symptom	*Primum defects*	*Secundum defects with pulm. hyperten.*	*Secundum defects without signific. hyperten.*	*Total*
Congestive cardiac failure	3	1	3	7
Dyspnoea and tiredness on exertion ..	2	3	18	23
Probable paroxysmal rhythm disturbances	0	2	2	4
Haemoptysis ..	0	1	1	2
Asymptomatic ..	1	0	8	9

* The very poor prognosis in these lesions is commented on by Wakai and Edwards,[5] in whose series of patients with common atrioventricular canal, 21 of 25 died before 6 years of age and only 2 reached adult years.

defects. Of the patients with secundum defects 8 were completely asymptomatic. Haemoptysis was experienced by 2 patients, one with pulmonary hypertension and one in congestive cardiac failure. Symptoms suggestive of attacks of paroxysmal tachycardia were present in 3 patients and 1 patient had several bouts of paroxysmal ventricular tachycardia and died from ventricular fibrillation. Sweating was occasionally noted in the younger patients. The symptoms are tabulated in Table III. The significance of dyspnoea and tiredness is often difficult to assess, because in some cases the patients have become cardiac neurotics.

Electrocardiogram

For many years an ECG pattern of so-called incomplete right bundle-branch block has been regarded as usual in atrial septal defects. Cabrera and Monroy[7] referred to this pattern, in which right precordial leads showed an rsR′ or rsR′s′ complex, as diastolic overload of the right ventricle. The failure of this pattern to disappear immediately after successful closure of the atrial septal defect seemed to disprove the 'diastolic overload' concept.

On the basis of the findings in our cases, we believe that this pattern is produced by dilatation of the right ventricular outflow tract. This area of the heart is undoubtedly dilated in cases of atrial septal defect and we believe this dilatation to persist or only slowly to disappear after corrective surgery. If this is so, it is possible that, with the relief of the shunt, the right ventricle may be able to empty before the left ventricle. In 1 patient, aged 44 years, this is suggested by the presence of 'paradoxical splitting' of the 2nd heart sound soon after closure of the defect (Fig. 1).

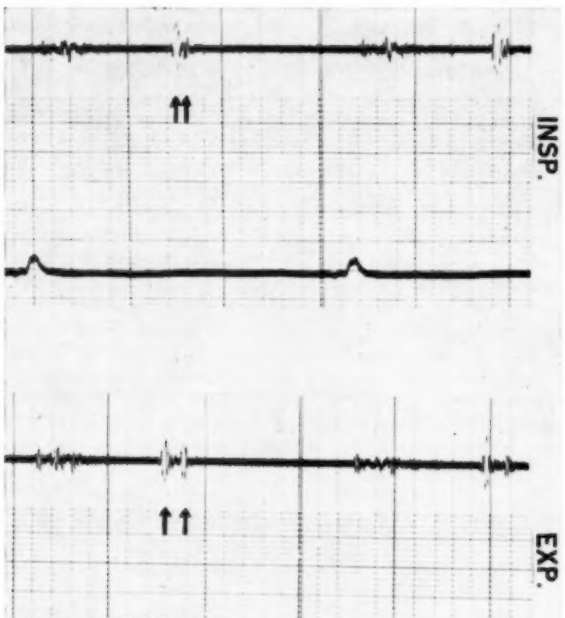

Fig. 1. Phonocardiogram showing paradoxical splitting of the 2nd heart sound after closure of an atrial septal defect.

In patients with pulmonary hypertension, a greater degree of hypertrophy of right ventricular muscle may be present and the ECG features may be somewhat different.

In our series, we recognize the following patterns of right ventricular preponderance in right chest leads (Fig. 2):

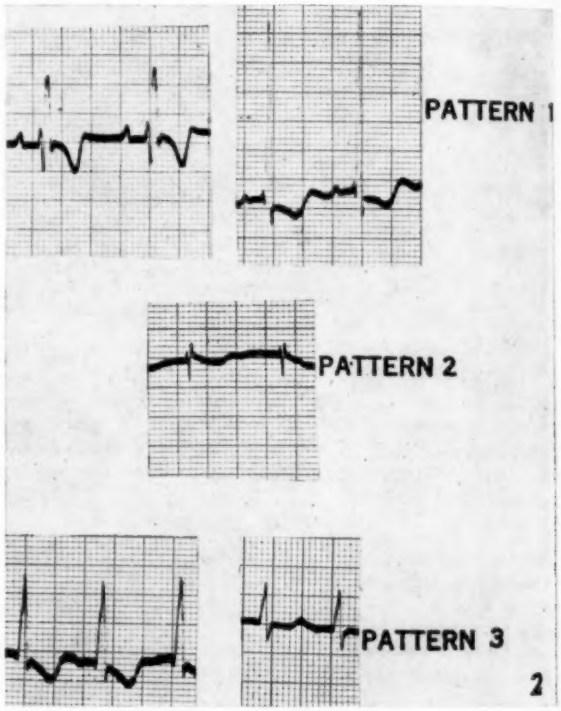

Fig. 2. The 3 basic patterns of the electrocardiogram in atrial septal defect. All tracings are of lead VI. The right-hand tracing in Pattern 1 is from a patient with severe pulmonary hypertension.

1. rsR′ or rsR′s′ where R′ was greater than twice the amplitude of s or s′.
2. rsr′ or rsr′s′ where r′ was less than twice the amplitude of s or s′.
3. qR or Rs or R.

Pattern 1 was present in 28 patients, of whom 19 were cases of secundum defect without pulmonary hypertension, 5 of secundum defect with pulmonary systolic pressures above 40 mm. Hg, and 4 of primum defect. In 18 of the 19 secundum defects without pulmonary hypertension the duration of the initial r was greater than 0·025 seconds while in 4 of the 5 with pulmonary hypertension the duration was less than 0·025 seconds.

Pattern 2 was present in 6 patients, 5 being cases of secundum defect and 1 of primum defect. In 2 of the 5 with secundum defects, the left-to-right shunt was large, while in 3 it was small as measured by cardiac catheterization

or presumed (in 1) by virtue of the small size of the defect found at necropsy.

Pattern 3 was present in 8 patients, all cases of secundum defect, 7 of whom were believed or known to have pulmonary hypertension.

In 1 patient no right ventricular 'preponderance' was found and it is believed that a 'balanced ventricular pattern' was produced by combined ventricular enlargement. This patient had an unusual form of 'septum primum' defect and will be reported separately.

Thus, while we have been unable to correlate a typical ECG pattern with pulmonary artery pressure, pulmonary flow, or pulmonary vascular resistance, we believe that careful appraisal of the ECG enables the type of case to be fairly accurately classified.

ECG evidence of right atrial enlargement was a common feature, being present in 38 (88%) of our cases. Left atrial enlargement was present in 3 cases, 1 of a primum defect, 1 associated with systemic hypertension, and 1 in severe congestive cardiac failure.

Evidence of left ventricular enlargement and the presence of left axis deviation constituted the most important single evidence of the defect being of the primum type. Thus, of 6 cases of primum defect, 5 showed left ventricular enlargement and the remaining 1, already referred to, was believed to have a 'balanced electrocardiogram'. One patient with a secundum defect showed left ventricular enlargement, attributable to systemic hypertension.

Radiological Features

In past years the detection of a so-called 'hilar dance' on fluoroscopy was regarded as an important feature of atrial septal defect. In our series, while pulsation in the main pulmonary arteries was often vigorous, a true 'hilar dance' was seldom present, and we pay little attention to this finding.

Roentgen films were taken in the customary views, namely postero-anterior, left and right anterior oblique, and lateral. With few exceptions, we have obtained all the information we require from routine PA films alone. However, we believe that none of these views are quite satisfactory for determination of right atrial enlargement. In view of the lateral situation of the right atrium and its tendency to enlarge in an anterior direction, we feel that examination in a slightly left anterior oblique position (about 10° rotation) will be advantageous; we are at present studying the value of this view. The following radiological features were analysed:

1. Size of the heart, as assessed by the cardiothoracic ratio.

2. Right atrial enlargement, assessed in the PA view and divided into 4 grades, viz. grade 1 (slight enlargement), grade 2 (moderate enlargement), grade 3 (marked enlargement), and grade 4 (aneurysmal enlargement).

3. Prominence of the right pulmonary artery on the one hand and main and left pulmonary arteries on the other. This is graded as grade 1, 2 or 3 for slight, moderate or marked prominence.

4. Pulmonary plethora, and particularly the visibility of large 'end-on' vessels well out in the lung fields. It is graded as grade 1, 2 or 3 for slight, moderate or marked plethora.

5. The so-called 'pulmonary hypertension picture', with sudden attentuation of pulmonary arteries towards the lung periphery.

6. Right and left ventricular enlargement.

Detailed Analysis

1. *Size of heart.* The cardiothoracic ratio was 53% or more in 25 cases. In general, those with primum defects had larger hearts (CTR over 60%), although the two largest hearts were in patients with secundum defects, one being in intractable heart failure, and the other having marked pulmonary hypertension. A normal-sized heart may be present even with large left-to-right shunts, and in 6 patients the CTR was under 50%, although the pulmonary flow was twice or more the systemic flow. The average CTR was 55%.

2. *Right atrial enlargement* of greater or lesser degree was always present (Fig. 3) and, in one patient in intractable heart failure, the right atrium was aneurysmal. The average grade of right atrial enlargement was 2·4, with no difference between primum and secundum defects.

3. *Prominence of pulmonary artery.* Because of 'overlapping' of the main and left pulmonary arterial shadows in the PA projection, these were analysed together; they were prominent in every case. The average grade of prominence was 2·5, with no difference between primum and secundum defects. Prominence of the right pulmonary artery was a feature, and is felt to be more notable than in other types of congenital heart disease with left-to-right shunt (Fig. 3). The average grade of prominence was 2·3, with no difference between primum and secundum defects.

4. *Pulmonary plethora.* Contrary to popular opinion,

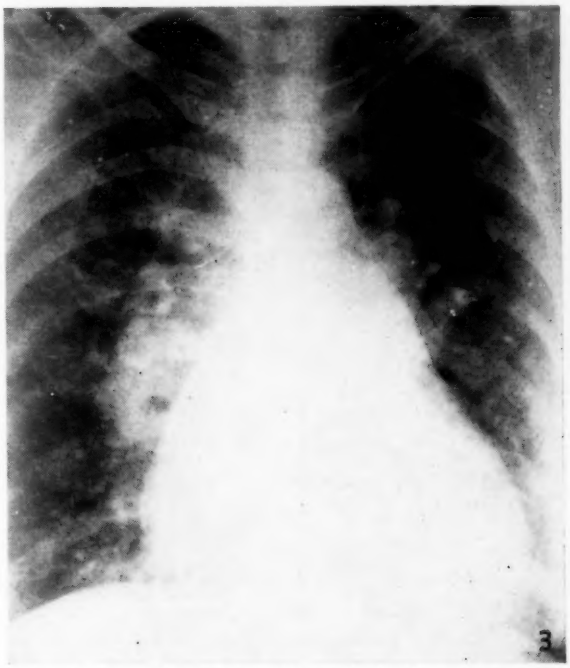

Fig. 3. Posterior-anterior radiograph showing enlargement of right atrium, both main pulmonary arteries, and right ventricle.

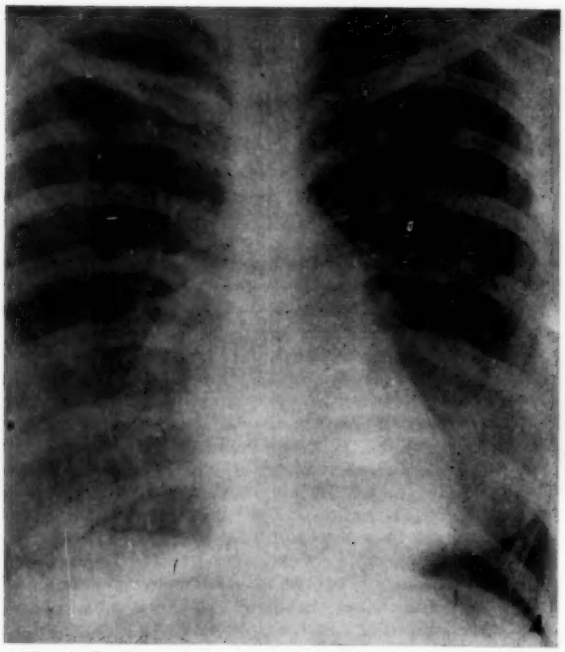

Fig. 4. Radiograph showing minimal 'plethora' of the lungs.

this was not a notable feature in the majority of our cases, the average grade being only 1·3 (Fig. 4). Since in most cases of atrial septal defect the pulmonary-artery pressure is normal or only very slightly elevated, the pulmonary vascular resistance must be diminished. This will allow a greater rate of flow through the lungs, and pulmonary blood volume may therefore not be increased proportionately to the increased flow. Much of the increased volume, too, will be taken up in the arteriolar and capillary bed. Hence, marked plethora would not occur. However, were the pulmonary-artery pressure to be moderately elevated, these factors would not apply, and pulmonary plethora would be more marked. Moreover, at low pressures, as exist in the pulmonary circuit, arterial distensibility increases strik-

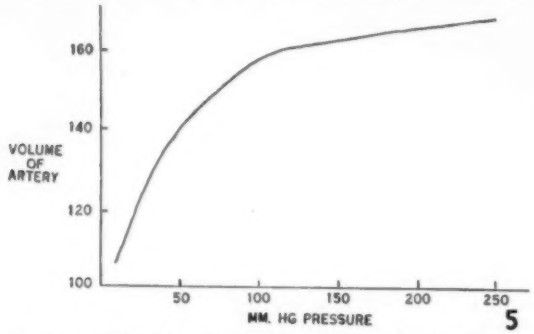

Fig. 5. Relationship of arterial volume to blood pressure.

ingly with increased pressure (Fig. 5). Thus, in those patients with pulmonary-artery systolic pressure above 40 mm. Hg, the average grade of plethora was 2·1 (Fig. 6).

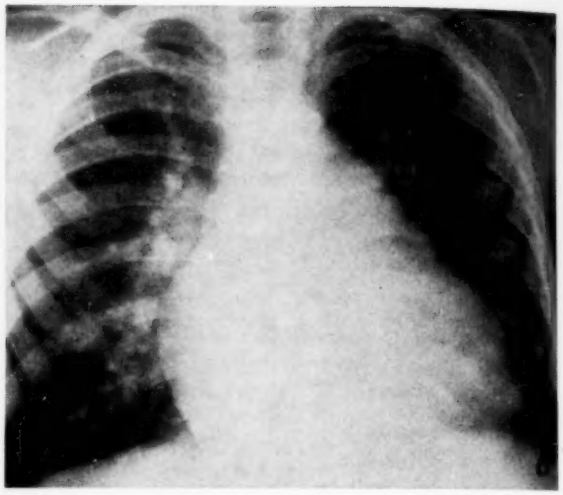

Fig. 6. Radiograph showing well-marked pulmonary plethora in a patient with moderate pulmonary hypertension.

5. '*Pulmonary hypertension picture*'. The striking attenuation of peripheral pulmonary arteries characteristic of pulmonary hypertension was noted in 8 patients (Fig. 7).

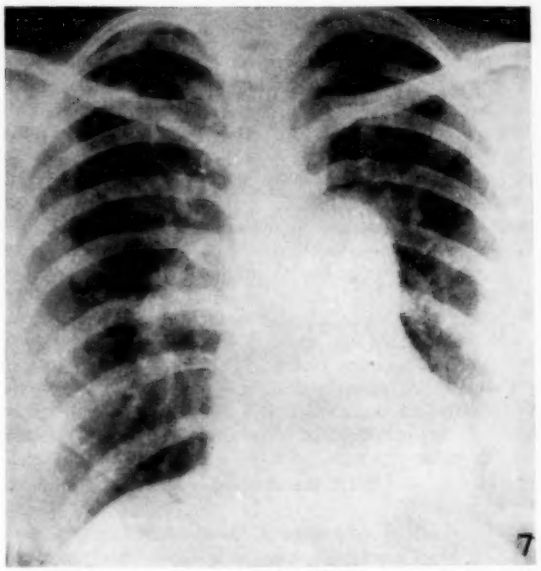

Fig. 7. Radiograph showing striking attenuation of peripheral pulmonary arteries in a patient with severe pulmonary hypertension.

In 3 of them pulmonary systolic pressure was over 65 mm. Hg, in 1 it was 45, and in the other 4 (all infants) cardiac catheterization was not performed. Pulmonary 'wedge angiography' showed a typical 'leafless tree' appearance in these cases (Fig. 8).

6. *Ventricular enlargement* was noted on the right side in most cases, usually of moderate degree, but marked in

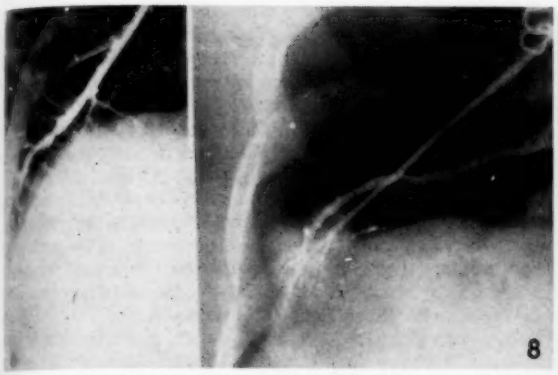

Fig. 8. Pulmonary 'wedge' angiographs (left) in a patient with severe pulmonary hypertension and (right) in a patient with normal pulmonary arterial pressure.

those in congestive heart failure. Left ventricular enlargement was difficult to assess, but was felt to be definite in 4 patients, all with primum defects.

Phonocardiography

This procedure was a valuable auxiliary to auscultation, particularly for determining the degree of splitting of the 2nd heart sound and the presence or absence of an ejection click (Fig. 9).

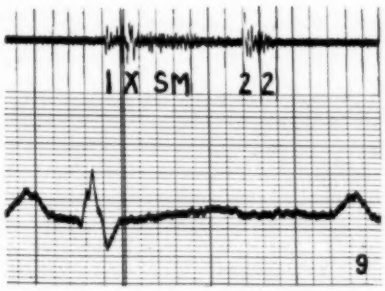

Fig. 9. Phonocardiogram demonstrating systolic ejection click.

Cardiac Catheterization

Cardiac catheterization was performed in 28 cases, by the usual techniques, the catheter being introduced *via* a vein in an arm, usually the left arm. In 16 cases the catheterization was personally performed by the authors, and in 13 of these the catheter could be passed *via* the septal defect into the left atrium (Fig. 10). Of the 3 cases where we did not enter the left atrium, 2 were *not* thought clinically to be atrial septal defects and exploration was not thorough, while in the 3rd a paroxysm of atrial tachycardia precluded exploration of the septum.*

The following features were analysed from data obtained at cardiac catheterization:

1. Pulmonary-artery pressure (mm. Hg).
2. Pulmonary arterial resistance; arbitrary units, viz.

* Since this paper was presented we have catheterized 12 more patients *via* the left arm and in each case we have been able to introduce the catheter into the left atrium and frequently into the left ventricle.

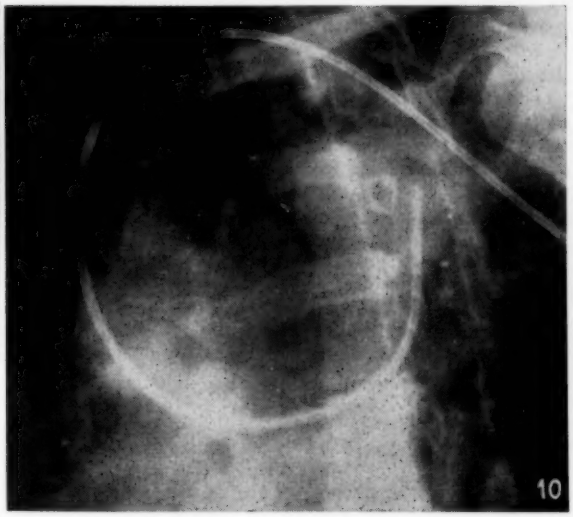

Fig. 10. Left anterior-oblique radiograph showing cardiac catheter in a pulmonary vein, *via* the atrial septal defect and left atrium.

$\frac{\text{MPA - MPCP,}}{\text{CO}}$ where MPA = mean pulmonary arterial pressure, MPCP = mean pulmonary 'capillary' pressure or 'wedge' pressure, and CO = pulmonary blood flow in litres per minute.

3. Degree of left-to-right shunt expressed as the ratio shunt flow: pulmonary flow.
4. A pressure gradient across the pulmonic valve.
5. Right atrial mean pressure (mm. Hg).

The haemodynamic data are summarized in Table IV.

TABLE IV. HAEMODYNAMIC DATA

Type of defect	Pulm. art. press. (mm. Hg)*			R. atrial press. (mean) mm. Hg*	Shunt flow/ pulm. flow	Pulm. art. resist-ance (units)	Pressure gradient at pulm. valve mm. Hg
	Sys.	Dias.	Mean				
S	38	22	30	8	0·5	4·0	0
S†	56	40	48	8	0·81	2·5	0
S	35	15	21	4	0·5	2·0	0
P	50	25	38	7	0·5	10·0	0
S	35	5	20	2	0·8	1·4	3
S	102	50	66	7	0·44	29·0	0
S	25	3	10	0	0·30	2·0	0
S	30	12	19	4	0·8	0·4	12
S	30	12	20	2	0·8	1·0	0
P	52	35	40	4	0·7	4·0	0
P	25	8	14	5	0·65	1·5	20
S	25	8	15	5	0·64	0·7	0
P	35	15	25	5	0·88	1·5	0
S	25	10	14	7	0·7	1·0	0
S	38	13	25	5	0·73	5·0	0
S	60	‡	‡	4	‡	‡	‡
P	70	20	42	0	0·7	16·0	0
S	30	10	18	1	0·8	1·1	0
S	23	8	15	2	0·61	2·0	0
S	28	10	17	0	0·7	1·6	0
S	50	22	35	5	0·7	7·0	5
S	45	20	30	12	0·74	1·8	0
S	38	10	20	4	0·9	0·4	0
S	38	16	24	4	0·57	2·0	0
P	30	14	20	3	0·8	1·6	0
S	28	7	17	5	0·65	1·4	0
S	65	35	48	4	0·7	8·0	0
S	28	5	15	3	0·6	3·0	8

S — Secundum. P — Primum.

* Reference point for all pressures is mid-axillary line.

† Plus mitral incompetence.

‡ Pulmonary artery not entered.

1. *Pulmonary-artery pressure.* Pulmonary-artery systolic pressure above 40 mm. Hg was found in 6 of 22 secundum defects (27%) and in 3 of 6 primum defects (50%). In 2 patients the pulmonary-artery systolic pressure was equal to systemic systolic pressure, measured simultaneously, and both patients showed well-marked right-to-left shunt at atrial level. The ages of the patients with pulmonary hypertension ranged from 2½ years to 39 years. Repeat catheterization was performed in 2 of the patients with pulmonary hypertension. In 1, a second study 1 year after the first showed a rise of systolic pressure from 65 to 73 mm. Hg. In the other, corrective surgery was performed 3 years after catheterization had shown a systolic pressure of 50 mm. Hg. As she failed to improve after complete closure of the defect, repeat catheterization was performed, which showed a systolic pressure of 75 mm. Hg. There is thus evidence that pulmonary hypertension in patients with atrial septal defect may be a rapidly progressive phenomenon. This evidence is further substantiated by clinical evidence in a 3rd patient, with the highest pressure in our series (systolic 102 mm. Hg); 6 months before catheterization, the pulmonic element of the 2nd heart sound was only moderately accentuated, but at the time of catheterization it was extremely loud.

2. *Pulmonary arterial resistance* was above 5 units in 5 patients, all with well-marked pulmonary hypertension, and in one of them* it was extremely high (29 units). This patient had severe pulmonary hypertension, with well-marked bi-directional shunting of blood, clinical cyanosis, and clubbing of the fingers. There was no direct correlation between pulmonary resistance and either pulmonary flow or pulmonary arterial pressure. However, all 5 patients with pulmonary arterial resistance above 5 units had mean pulmonary arterial pressures of more than 30 mm. Hg, whereas only 2 of 22 patients with pulmonary arterial resistance below 5 units had pressures above 30 mm. Hg (Fig. 11). There is, therefore, evidence that pulmonary

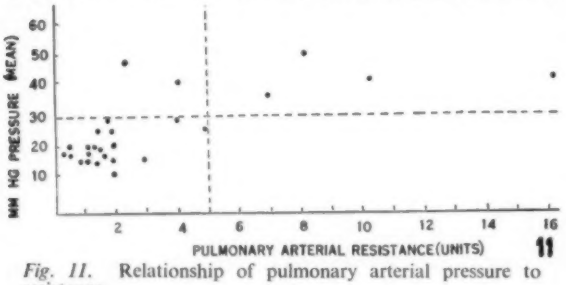

Fig. 11. Relationship of pulmonary arterial pressure to resistance.

hypertension is due to increased pulmonary arteriolar resistance. Paucity of material for histological examination does not permit us to say whether the increased pulmonary arteriolar resistance is due to organic vascular disease or simply increased tone.

3. *Degree of left-to-right shunt.* In all but 2 of the patients catheterized, shunt flow constituted 50% or more of pulmonary flow. In one of these 2 patients the defect was regarded as being small, while in the other, in whom operation confirmed a large defect, the small left-to-right shunt was attributable to severe pulmonary hypertension.

* Not shown in Fig. 11.

4. *A pressure gradient across the pulmonary valve* was found in 5 patients, ranging from 3 to 20 mm. Hg. The degree of gradient was not proportional to the amount of shunt or total pulmonary-artery flow.

5. *Right atrial mean pressure* higher than 5 mm. Hg was found in 6 patients, 5 of whom showed clinical evidence of congestive cardiac failure. In those patients the average pulmonary-artery systolic pressure was 53 mm. Hg, while in those whose mean atrial pressure was less than 5 mm. Hg the average pulmonary artery systolic pressure was 37 mm. Hg. However, 2 of the 6 with elevated right atrial pressure had normal pulmonary-artery pressures, while 5 of 22 with normal right atrial pressures had pulmonary hypertension. In those cases where the left atrium was entered, the left atrial mean pressure was found to be equal to the right atrial pressure, or up to 3 mm. higher. Anomalous entry of a pulmonary vein into the right atrium was shown to be present in only 1 patient in our series; it was not diagnosed pre-operatively.

TREATMENT

Seventeen patients in the present series were subjected to surgical treatment for closure of the septal defect, 1 by a 'closed' technique, and 16 by open-heart surgery with either hypothermia or extracorporeal circulation.*

Four of the 17 patients died. They comprised one patient with a primum defect who was subjected to surgery against our advice and who died after closure by a closed technique; one patient with a complete common atrioventricular canal who died immediately after repair under extracorporeal circulation; one patient in gross congestive heart failure who died from haemorrhage due to a bleeding diathesis several hours after closure under extracorporeal circulation; and one patient with equalization of pulmonary and systemic blood pressures and well-marked bi-directional shunt, who died several hours after closure of the defect under hypothermia.

Of the 13 survivors, in all but one the result has been good, with a follow-up of 3 - 16 months. The one patient who failed to improve was found at re-catheterization to have persistent pulmonary hypertension but no residual shunt.

DISCUSSION

Atrial septal defect is a common congenital cardiac anomaly, the diagnosis of which is usually possible on clinical grounds and can be confirmed by cardiac catheterization. In early infancy the clinical picture is atypical, and the anomaly should be considered in all infants with evidence of heart disease.

The mortality rate in infancy is high. In the patients who survive infancy, the presence of primum defect indicates a persistently poor prognosis, and so does the advent of pulmonary hypertension in those with secundum defects. Even in those with secundum defects and normal pulmonary-artery pressures, the life span is limited and cardiac failure may develop in the 4th and 5th decades of life. The surgical repair of secundum defect in patients without pulmonary hypertension or cardiac failure has an extremely low mortality,[4] but once these two complications develop the risks of surgery increase alarmingly.[5] For these reasons, operation should be advised *on diagnosis* in uncomplicated secundum

* See article by Mr. D. Fuller, at page 797 of this issue, which deals with the 16 cases operated on by 'open' technique.

defects. There is no justification for awaiting the development of symptoms. As surgery produces an anatomical cure, there is every reason to predict a normal life span in these cases.

The risks of surgery are significantly greater in primum defects, but the natural history of the disease is such as to warrant facing this higher risk.

We believe, in conclusion, that atrial septal defect should be likened to patent ductus arteriosus and that early diagnosis and early treatment are strongly indicated.

SUMMARY

The clinical, electrocardiographic, phonocardiographic, radiological and haemodynamic features in 43 cases of atrial septal defect are described. The prognosis of the disease is discussed and the safety of surgery in uncomplicated cases is stressed. A plea is made for early diagnosis and treatment of this defect.

Several of our patients were investigated at the Cardiac Clinic at the Johannesburg General Hospital and we thank Prof. G. A. Elliott for allowing us access to their files. We thank Dr. J. Meyer and Dr. W. Scott for their assistance at cardiac catheterization, and the Photographic Unit, Department of Medicine, University of the Witwatersrand, for reproduction of the figures. We are greatly indebted to Mr. David Adler and Mr. Denis Fuller for their cooperation and for their encouragement and advice at all times.

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VRAE BEANTWOORD : QUESTIONS ANSWERED

POLIOMYELITIS BOOSTER DOSES

Q—*Thousands of children have had poliomyelitis vaccine by injection. Has any decision been reached yet on the question of booster doses 2 or 3 years after the last injection of a 3- or 4-injection course? Could this be given by the oral route when the oral vaccine becomes available?*

A—It is now generally accepted that within 2 years from the time of the third injection of Salk vaccine, a fourth, or 'booster' dose should be administered.

Serum-antibody assays in adolescents and adults reveal that for all 3 types of virus there is a fall of between 78 - 81% in the antibody levels within 18 - 24 months following such a booster dose.[1] Anyone subject to a special risk of contracting poliomyelitis should have the booster dose sooner than 2 years after the last injection.

The immunity produced by the use of Salk vaccine is of the passive variety and, as far as we know, wanes with the passage of time. Another drawback is that while in approximately 90 - 95% of cases it protects against paralytic and meningeal complications, it does not render the alimentary tract insusceptible to invasion following naturally occurring infection. While this might not be of much importance to the immunized subject, multiplication of virus in his intestinal tract and its subsequent excretion does nothing to block the main avenue of the spread of infection.

The use of live-attenuated vaccine of the Sabin type produces an extremely mild attack of poliomyelitis with occasional nausea and diarrhoea and, in the process, active immunity. The intestinal tract, after the active immunization, is no longer receptive to virus in the natural form so that individuals who have had live vaccine, while being completely protected themselves, do not become 'carriers', nor do they excrete the virus. From the public health point of view this is of the utmost importance in our attempts to control this disease.

In the administration of live virus, it is considered essential that, for successful 'takes' against all types of poliomyelitis virus, it should preferably be administered during the winter months when the many mild forms of other enteroviruses are at a minimum in the intestinal tract. It is also suggested that 3 doses of the triple vaccine, containing types I, II and III, be administered orally with a month's interval between the doses. By this means it is anticipated that 95% of 'takes' for each type should be accomplished. From the above, it can be accepted that the use of live-attenuated virus vaccine in spaced doses can be used in substitution for the booster dose of Salk vaccine, thereby converting a passive into an active immunity.

1. Kendall, E. J. C. *et al.* (1960): Brit. Med. J., **1**, 1689.

Readers are invited to submit questions for this column to the Editor, *South African Medical Journal*, P.O. Box 643, Cape Town.

UNITED STATES PUBLIC HEALTH SERVICE INTERNATIONAL POSTDOCTORAL RESEARCH FELLOWSHIPS: INFORMATION STATEMENT

The development of research in medicine and biology has been strengthened in the United States by periods of study and research spent by citizens of this country in the laboratories of other countries. For many years the National Institutes of Health of the Public Health Service has helped to finance this productive interchange. It has now become possible to extend the US Public Health Service fellowship programme through the award of a limited number of fellowships to highly qualified scientists from outside the United States to work in the medical and biological laboratories of the USA.

The research training of these scientists from other countries will be financed to strengthen medical research throughout the world. It will give these outstanding scientists in health-related fields a chance to share knowledge with kindred groups by conducting research in the United States. The fellows, as well as the host laboratories, should benefit from this exchange of knowledge and techniques.

The National Institutes of Health intends to administer this programme in a manner which will make the work of the scientists from abroad most productive. Experience has shown that an essential part of this process is a clear understanding of the terms and conditions under which the fellowships are awarded. These terms and conditions, which have been developed with the advice of scientists from the other countries, are outlined below.

One particularly important condition of the awards, noted below in Part 4, is that the fellows return to their home countries.

1. *Method of Applying*

Appropriate national research organizations have been authorized to nominate fellows. These organizations may

obtain application forms, together with necessary instructions and information, from the National Institutes of Health. The secretary of a national research organization should send to the National Institutes of Health only completed applications which the organization has formally endorsed as meeting the conditions set down in the following section.

2. *Minimum Requirements*

The candidate must have:

(*a*) Obtained a doctoral degree or its equivalent in one of the medical or related sciences and also have demonstrated outstanding research promise.

(*b*) Made satisfactory arrangements with a laboratory in the United States at which he proposes to train. (Such arrangements are often made through correspondence between a senior scientist and a counterpart in the United States where a suitable research opportunity or training in the applicant's field of interest can be provided. Copies of such correspondence should accompany the application.) A Facilities and Commitment Statement blank for this purpose will be forwarded in each application packet.

(*c*) Acquired a workable reading and speaking knowledge of the English language.

(*d*) Demonstrated proficiency in research, with indication that he will pursue a research and/or academic career for a reasonable period after training.

(*e*) Met such standards as are required by his own national research organization.

3. *Period and Terms of Award*

(*a*) *Period of award.* An award will usually be made for 1 year, but a second year or part thereof may be approved when adequately justified. Application for extension must be made through the committee which made the original nomination. Nominations for extension will constitute one of the allowable nominations from that country and not an additional one. Final approval will be by the committee at the National Institutes of Health.

(*b*) *Stipend and allowances.* (1) A stipend of $4,500 is provided — in addition, $500 is provided for the spouse and each dependent child, whether or not the dependents accompany the fellow to the United States; (2) Travel for the fellow only (not for dependents) is provided at the rate of 8 cents per mile from home to laboratory in the United States and return; and (3) No allowance will be made for shipment of personal or household effects.

(*c*) *Beginning dates.* The fellow may commence training at any date which he and his laboratory of choice find convenient, within 10 months of formal notification of award.

(*d*) *Report of activities.* A brief written report of scientific activities shall be prepared by the fellow at the end of his visit, to permit evaluation of scientific utility of the foreign fellowship programme.

4. *Passports and Visas*

Fellows will be responsible for obtaining passports and visas required for entrance into the United States. All fellowship awards are made subject to ability of the applicant to obtain the necessary papers for entry into the United States. A letter of appointment and a letter authorizing the use of the exchange-visitor number for obtaining an exchange-visitor's visa are provided by the National Institutes of Health to successful applicants. *This type of visa requires that the fellow return to his homeland after completion of training.*

5. *Visit to the National Institutes of Health*

All fellows will be welcome to visit the National Institutes of Health for a brief period. The Foreign Grants and Awards office should be notified of intent to visit at least 1 month in advance.

6. *General Information*

(*a*) *Payment of stipends.* Pay begins upon the fellow's arrival in the United States. The sponsoring institution will have been given a grant from which the fellow will receive funds to cover his stipend, dependency allowances, travel, and laboratory expenses. If emergency funds are required early in the fellowship tenure, the sponsoring laboratory or institution may make such funds available, and this advance will be deducted gradually from subsequent stipends.

(*b*) *Laboratory expenses.* An amount of $500 for each year of the fellowship will be provided as a part of the grant to the institution. These funds may be used for any purpose which the institution concludes will further the fellow's research training, including transportation costs and *per diem* allowance to permit attendance at a scientific meeting within the United States.

(*c*) *Income tax.* Stipends and dependency allowances up to $300 per month paid for this programme will normally not be declarable as income under the US Federal Income Tax Law, although an individual tax return must be filed annually. Fellows are urged to consult university officials on this matter shortly after arrival.

(*d*) *Health and accident insurance.* Health and accident insurance will be required of all fellows through a group policy maintained by certain United States Government agencies for non-citizens who are in the United States for training. The premium for such insurance is set annually. The current insurance rates will be quoted at the time of the award. The premium will be advanced from grant funds but the grant will be reimbursed through deductions from pay checks to the fellow.

(*e*) *Special information and assistance.* If any difficulties arise after arrival in the United States, or if further information is required, the fellow may communicate with the Chief, Foreign Grants and Awards, Division of General Medical Sciences, National Institutes of Health, Bethesda 14, Maryland. (telephone OLiver 6-4000, extension 4335, or WHitehall 6-3231). The fellow will be assisted in any way possible.

South Africa

Address inquiries to: Mr. C. G. Hide, Science Cooperation Division, Information and Special Services Department, CSIR, P.O. Box 395, Pretoria.

Applications must reach the above address before 5 October, 1960.

OPKNAPPINGSKURSUS : NAGRAADSE SKOOL BEPLANNINGSKOMITEE, BLOEMFONTEIN

Die volgende opknappingskursus van die Nagraadse Beplanningskomitee, Bloemfontein, sal vanaf 23 tot 25 September 1960 gehou word. Die program is soos volg:

Vrydag 23 September

2 - 2.45 nm.	‚Stress alarm and the general adaptation syndrome.' 'n Oorsig en waardering van Selye se benadering tot die verskynsels van gesondheid en siekte: Dr. A. G. M. Morrison.
2.45 - 3 nm.	Pouse.
3 - 3.45 nm.	Hipotiroïdisme: Dr. G. W. Snyman.
3.45 - 4.15 nm.	Teepouse.
4.15 - 5 nm.	Swaminfeksies van die huid: Dr. I. J. Venter.
5 nm.	Pouse.
8 - 10 nm.	(i) Vaskulêre fistels. Teepouse. (ii) Indrukke i.v.m. Amerikaanse geneeskunde na aanleiding van 'n onlangse besoek aan die V.S.A.: Prof. A. J. Brink.

Saterdag 24 September

9 - 9.45 vm.	Behandeling van 'n paar algemene frakture en ontwrigtinge: Dr. L. Muller.
9.45 - 10.15 vm.	Teepouse.
10.15 - 11 vm.	Organiese psigose: Dr. A. J. van Wyk.
11 - 11.15 vm.	Pouse.
11.15 - 12 nm.	‚Fibrocystic disease of pancreas.': Dr. P. Fischer.
12 nm.	Pouse.

2.30 - 4.30 nm.	Filmvertoning en referate onder leiding van professor Geldenhuys.
8 nm.	Sosiale funksie.

Sondag 25 September

9 - 10 vm.	Demonstrasie van kliniese gevalle onder toesig van dr. F. P. Scott.
10 - 10.15 vm.	Teepouse.
10.15 - 11.15 vm.	Demonstrasie van kliniese gevalle onder toesig van dr. F. P. Scott.

Praktisyns wat in die kursus belangstel, word uitgenooi om so spoedig moontlik met die Eresekretaris, Posbus 834, Bloemfontein, in verbinding te tree.

IN DIE VERBYGAAN : PASSING EVENTS

South African Institute for Medical Research, Johannesburg. Staff Scientific Meeting. The next meeting will be held on Monday 26 September at 5.10 p.m. in the Institute Lecture Theatre. Dr. P. A. D. Winter, of the Polio Laboratory, Rietfontein, Johannesburg, will speak on 'Live poliovirus vaccination'.

* * *

Correction. In an Editorial entitled 'Tapeworm infestation' which appeared in the issue of the *Journal* for 13 August 1960 (**34**, 685) the brand name of dichlorophen was given in brackets as 'Dicestrol', when in fact it should have read 'Dicestal'. 'Dicestal' is the name given to a veterinary preparation of dichlorophen, manufactured by Maybaker (S.A.) (Pty.) Ltd. and indicated for the treatment of tapeworm infestation in dogs and cats. This firm produces a more purified form of dichlorophen for human use, under the brand name of 'Antiphen'.

IN MEMORIAM

SELIG ABRAHAM LANGE, M.B., CH.B. (CAPE TOWN)

Dr. B. Isaacson, of Cape Town, writes:

In the passing of Dr. Selig Lange, Cape Town has lost the finest example of what is now called the 'personal doctor'. He had that 'something else' that gave his patients comfort and assurance by his mere presence alone.

Dr. Lange

Born in Senekal, he received his early education in Berlin, but he returned to this country at the age of 18. He studied metallurgy and then turned to medicine, probably because he preferred to work with human beings for whom he had such love and compassion. For this profession he was eminently suited. He was one of the early graduates of the University of Cape Town.

He travelled extensively and, wherever he went, London, Vienna, and America, he visited the hospitals so as to keep in touch with recent advances in medicine. Member of the Race Relations Society, subscriber to the orchestral concerts, and to the Botanical Society of South Africa, he was a man of wide interests and a patron of all humanitarian movements. His interest in art, literature, and music — the latter was a passion with him, he was listening to a Beethoven symphony the night before he died — was shared by his wife Felicity Friedlander, his retiring and gentle companion.

In spite of long ill-health he had a great zest for life and he enjoyed to the full the wonder and beauty of nature which he tried to capture in his photographic expeditions. He was in the habit of walking with a group of friends on Saturday afternoons, enjoying the profusion of trees and plants in the lovely and less frequented spots of the Cape.

But it was perhaps in his personal relationships that he was most remarkable, bringing hope and comfort to the sick and cultivating the tender plant of friendship among his numerous friends with such warmth and affection. He corresponded for years with friends he had made during the war, young men from distant countries who received his hospitality and friendship with gratitude.

The Salesian Institute, to which he gave years of devoted service, has inscribed a plaque to his memory and has named a wing of the building after him. St. Joseph's Home for Crippled Children, where he served for 26 years, is also naming a wing after him.

Our sincere sympathy goes out to his wife, his only son Paul, and his nearest relations.

Those who knew him will not forget him.

Dr. W. E. Laufer, writes:

Dr. Selig Lange, who died suddenly in Cape Town on 27 July, had been in poor health for some months. He was born in the Orange Free State Republic, but spent much of his early life in Europe. When he returned to this country he at first worked for some years as a geologist, but then proceeded to the University of Cape Town to study medicine. He qualified M.B., Ch.B. in 1926, and then went to Europe where he did several years' postgraduate work in Vienna, Berlin, and London. He started in general practice at Arlington, OFS, and then came to Claremont, Cape, where he practised until his death.

Selig Lange, whom I knew for 22 years, embodied all those qualities one seeks and admires in a good doctor and a good man. In his work he was painstaking and conscientious, and his professional ability was well above average. He knew people as well as diseases; this made him an acute observer and an astute diagnostician. He read widely and was ever ready to learn of the latest advances in medicine. Disarmingly humble, he was always pleased to give his less experienced colleagues the benefit of his wide knowledge. Dr. Lange had a very large practice, but in spite of this he never acquired sufficient of the world's goods; he was too much an idealist and a humanitarian to be a successful financier. Yet, of the little he had, he gave more than he could afford to various individuals in need and to charitable organizations, both in this country and overseas. His heart went out to any sufferer anywhere, and no one came to him in vain for help.

Dr. Lange had many and varied interests apart from his medical work. He was a devoted nature-lover, and had an encyclopaedic knowledge of plants and trees and geological formations. He was also widely read in the Arts and the Humanities, and possessed a well-stocked library of excellent books. An ardent music-lover, one of the few pleasures he permitted himself was to attend a weekly symphony concert. An interesting conversationalist and a good listener, he was at his best among a small circle of good friends and relatives. He detested gossip, and it was his philosophy that it is better to say nothing than to speak evil of one's fellows. Selig Lange always believed the best of everyone, and he knew how to bring out the best in people if this was not immediately apparent. Even though his experiences on occasion severely shook his faith in human nature, he did not alter his beliefs. In his consulting room there hung a framed poem which ended: 'Let me live in my house by the side of the road and be a friend of men.' And a friend of men he was, to the very last.

Although not orthodox, Dr. Lange was well-informed about his Jewish faith, and he had an intimate knowledge of Hebrew writers and of Jewish history. He was also a keen Zionist, who followed with avid interest the development of the Zionist movement, the establishment of the State of Israel, and its subsequent achievements.

Just as outstanding as his deep humanity, was Dr. Lange's quiet courage in adversity. He was plagued by ill-health throughout his life, one major illness following another in all too rapid succession. As often as he was struck down, however, he rebuilt his practice. Having latterly recovered from a major abdominal operation, and also suffering from chronic coronary insufficiency, he told me two months before his death that he was still not thinking of retiring, and I once again found him attempting to build up his practice. Never did he utter a word of complaint about his very many misfortunes.

Selig Lange will be mourned by hundreds of people, of all races and creeds, as a doctor and a friend whose life was an example and an inspiration to all of them. His memory will live for many years. Physically and mentally he was always at the service of all those who needed him. In all the years I have known him, whether he was healthy or ill, I cannot recall ever having seen him angry, or without his habitual friendly smile.

Dr. Lange married Miss Felicity Friedlander, who was his faithful companion and who stood by him devotedly in all his misfortunes. Their son Paul is a businessman in Cape Town. To Mrs. Lange and his son we tender our sincere condolences; we all miss him as much as they do, and we share their sorrow.

NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

WYNAL

Wyeth Laboratories (Pty.) Ltd., announce the introduction of Wynal Vaginal Inserts, for the complete treatment of vaginal infections, and supply the following information:

Wynal vaginal inserts are highly effective in the treatment of vaginitis caused by trichomonas, monilia, bacterial infection, or vaginitis of mixed aetiology.

Wynal inserts contain: bismuth glycolylarsanilate, for trichomonicidal action and effectiveness against fungi and bacteria; sulfathiazole, for action against bacterial infection; sodium lauryl sulfate, for detergent action to facilitate dispersal of the compound and penetrative power of the infecting matter; and also allantoin, for healing.

Formula. Each insert contains: Bismuth glycolylarsanilate 250 mg., sulfathiazole 500 mg., allantoin 10 mg., sodium lauryl sulfate 3 mg., lactose, dextrose, sucrose, magnesium stearate, and cornstarch c.s., in a hydrosoluble base which facilitates efficient liberation of the active ingredients.

Directions. One Wynal insert should be placed high in the vagina every other night for 10 - 14 days.

Wynal is supplied in bottles containing 12 inserts.

Further information may be obtained from Wyeth Laboratories (Pty.) Ltd., P.O. Box 42, Isando, Transvaal.

ORENZYME

Westdene Products (Pty.) Ltd. announce the introduction of Orenzyme and supply the following information:

Orenzyme is a new oral enzyme tablet containing the proteolytic enzymes trypsin, chymotrypsin and ribonuclease. The tablets are enterically coated to insure disintegration and release in the intestinal tract. Orenzyme is the first available oral enzyme preparation to provide systemic anti-inflammatory activity, and represents a distinct advance in the well-established field of trypsin (Parenzyme) therapy, for the resolution of inflammation and oedema.

Clinial effects in inflammatory disorders. Orenzyme removes fibrin deposits from capillaries and connective tissue; restores local circulation; hastens absorption of extravasated blood and tissue fluids; liquefies mucous secretions; promotes healing; and increases effectiveness of antibiotics.

Mode of action and rationale in inflammation. Parenzyme and Orenzyme hasten resolution of inflammation and oedema by enzymatic action on the fibrin deposits blocking the capillaries and tissue spaces. Normal permeability is restored, and circulation re-established. As a result, waste products are removed, and the multiple defence forces of the blood gain access to the inflammatory site, preventing further tissue damage and hastening the healing process.

Indications for Parenzyme and Orenzyme

(a) Inflammation associated with peripheral vascular disease: acute thrombophlebitis, chronic thrombophlebitis, phlebothrombosis.

(b) Ocular inflammation: extra-ocular trauma, iritis, vitreous haemorrhage, postoperative hyphema.

(c) Bronchopulmonary inflammation: to liquefy thick tenacious mucous exudates in severe bronchitis, bronchiectasis, emphysema, and bronchial asthma.

(d) Traumatic wounds: crush injuries, contusions, fractures, concussions, haematomas, sprains.

(e) Ulcerations: stasis, ischaemic, decubitus, diabetic.

(f) Postoperative tissue reactions following: oral surgery, plastic surgery, urologic surgery.

(g) Inflammatory skin disorders: herpes zoster, dermatitis, psoriasis, carbuncles and furuncles.

Advantages. Orenzyme is the first effective oral enzyme for systemic anti-inflammatory therapy. It is ideal for adjunctive/maintenance therapy following Parenzyme Aqueous. There are no known incompatabilities to Parenzyme and Orenzyme. Other specific medicaments such as anticoagulants and antibiotics may be given when indicated. For infections, appropriate antibacterial therapy should be used concurrently.

Side-effects. Both Parenzyme and Orenzyme have a wide margin of safety and are rarely contra-indicated. They should be used with caution in patients with abnormalities of the blood-clotting mechanism, such as haemophilia. Patients with severe renal or hepatic disease should be followed carefully during Parenzyme or Orenzyme therapy.

Dosage. For best results, adequate dosage is important. Initiate treatment with Parenzyme Aqueous: 1 c.c. (5 mg.) injected intramuscularly once or twice daily for several days, followed by Orenzyme, 1 tablet 4 times daily as indicated for maintenance. Infants and children up to 2 years: 0.2 c.c. (1 mg.). Older children: 0.5 c.c. (2.5 mg.).

or

Orenzyme: 2 tablets swallowed with a glass of water 4 times daily. Dosage may be reduced to 1 tablet 4 times daily once symptoms begin to disappear.

Orenzyme is supplied in bottles of 48 red enteric-coated tablets. Each oral tablet is equivalent in proteolytic activity to 20 mg. crystalline trypsin.

Orenzyme is marketed in South Africa by Mer-National Laboratories (Pty.) Ltd., P.O. Box 6742, Johannesburg, and distributed by Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

STELADEX

SKF Laboratories (Pty.) Ltd. introduce Steladex spansule capsules, and supply the following information:

Each Steladex spansule capsule contains 2 mg. trifluoperazine and 10 mg. dexedrine (dexamphetamine sulphate).

Indications. Steladex is used in the treatment of obesity, especially when psychoneurotic factors underlie the condition. Steladex alleviates the stress of dieting as well as curbing the appetite of the patient on a low-calorie diet. Steladex curbs the patient's appetite all day long with 1 morning dose. It suppresses any psychoneurotic factors underlying the obesity and leaves the patient composed but alert.

Contra-indications and side-effects: Steladex should be used with caution in the presence of severe hypertension, advanced cardiovascular disease, or extreme excitability. Side-effects are chiefly nervousness and insomnia, but these are infrequent, mild, and transitory.

Dosage. One Steladex spansule capsule daily, taken in the morning.

Steladex is supplied in containers of 30 sustained-release spansule capsules.

Further information may be obtained from SKF Laboratories (Pty.) Ltd., P.O. Box 38, Isando, Transvaal.